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OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE THIRTIETH

PHILADELPHIA
PRINTED FOR THE COLLEGE
1908

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The present volume of TRANSACTIONS contains the papers read before the College from January, 1908, to December, 1908, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WILLIAM ZENTMAYER.

CATALOGUED
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* Died May 17, 1908

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ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES*
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGER
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS†
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C. WOOD
1904	ARTHUR V. MEIGS
1907	JAMES TYSON

* Died four months after his election.
† Resigned on account of ill-health.

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FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

DECEMBER, 1908.

* Non-resident Fellows.
† Fellows who have commuted dues.

ELECTED

*1883. ABBOT, GRIFFITH E., Ph.D., M.D. Leominster, Mass.

1892. ABBOTT, ALEX. C., M.D., Sc. D. (Hon.), Professor of Hygiene and Bacteriology, and Director of the Laboratory of Hygiene in the University of Pennsylvania. 4229 Baltimore Ave.

1905. ADLER, LEWIS H., JR., M.D., Professor of Diseases of the Rectum in the Philadelphia Polyclinic and College for Graduates in Medicine; Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 1610 Arch St.

1903. ALLEN, ALFRED REGINALD, M.D. Lecturer on Neurological Electro-Therapeutics and Instructor in Neurology and Neuropathology in the University of Pennsylvania. 111 S. Twenty-first St.

1906. ALLEN, FRANCIS OLCOTT, JR., A.B., M.D., Surgeon to the Dispensary of the Presbyterian Hospital; Surgeon to the Dispensary of the Methodist Episcopal Hospital. 323 S. Sixteenth St.

1896. ALLYN, HERMAN B., M.D., Associate in Medicine in the University of Pennsylvania; Physician to the Philadelphia Hospital. 501 S. Forty-second St.

1888. ANDERS, JAMES M., M.D., LL.D., Professor of the Theory and Practice of Medicine and Clinical Medicine in the Medico-Chirurgical College; Consulting Physician to the Jewish Hospital Association, Philadelphia. 1605 Walnut St.

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ELECTED

1869. ANDREWS, T. HOLLINGSWORTH, M.D. 1119 Spruce St.

1905. ANSPACH, BROOKE M., M.D., Gynecologist and Obstetrician to the Philadelphia Hospital; Assistant Gynecologist to the University Hospital; Instructor in Gynecology in the University of Pennsylvania; Pathologist to the Kensington Hospital for Women. 1819 Chestnut St.

1905. APPLEMAN, LEIGHTON F., M.D., Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College; Instructor in Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmologist to the Frederick Douglass Memorial Hospital. 308 S. Sixteenth St.

1906. ASHHURST, ASTLEY P. C., A.B., M.D., Surgeon to the Dispensary of the Episcopal and of the Children's Hospitals; Assistant Surgeon to the Orthopædic Hospital; Chief of the Gynecological Dispensary of the Pennsylvania Hospital; Prosector to the Associate Professor of Applied Anatomy in the University of Pennsylvania. 2000 De Lancey Place.

1893. ASHTON, THOMAS G., M.D., Adjunct Professor of Medicine in the University of Pennsylvania; Physician to the Philadelphia Hospital. 1814 S. Rittenhouse Square.

1857. ATLEE, WALTER FRANKLIN, A.M., M.D., Corresponding Member of La Société des Sciences Médicales de Lyons; Consulting Surgeon to St. Luke's Hospital, Bethlehem; Visiting Physician to the Preston Retreat. 210 S. Thirteenth St.

1906. BABBITT, JAMES A., A.B. (Yale), A.M. (Haverford), M.D., Physical Director and Associate Professor of Physiology at Haverford College; Instructor in Diseases of the Nose and Throat in the Philadelphia Polyclinic; Laryngologist to the Dispensary of the Children's Hospital; Laryngologist and Aurist to the Home for the Training in Speech of Deaf Children before they are of School Age. 121 S. Eighteenth St.

1852. BACHE, THOMAS HEWSON, M.D. 233 S. Thirteenth St.

1903. BACON, JOHN, M.D. Torresdale, Pa.

†1892. BAKER, GEORGE FALES, B.S., M.D. 1818 Spruce St.

1889. BALDY, JOHN MONTGOMERY, M.D., Professor of Gynecology in the Philadelphia Polyclinic; Surgeon to the Gynecean Hospital; Consulting Surgeon to the Jewish and to the Frederick Douglass Memorial Hospitals. 2219 De Lancey Place.

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ELECTED

1898. BALLIET, TILGHMAN M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3709 Powelton Ave.

1883. BAUM, CHARLES, A.M., M.D., Ph.D. 1828 Wallace St.

1908. BEARDSLEY, EDWARD J. G., M.D., L.R.C.P. (Lond.), Demonstrator of Physical Diagnosis and Clinical Medicine in Jefferson Medical College; Physician to the Out-patient Medical Department of Jefferson Medical College Hospital; Physician to the Henry Phipps Institute; Examining Physician for the White Haven Sanatorium. 2030 Chestnut St.

1883. BEATES, HENRY, JR., M.D. 260 S. Sixteenth St.

1874. BENNETT, W. H., A.M., M.D., Physician-in-Charge of the Seashore House for Invalid Children, and of the Seaside House for Invalid Women, Atlantic City; formerly Physician to the Episcopal Hospital, and Physician-in-Charge of St. Christopher's Hospital for Children. 1837 Chestnut St.

1896. BEYEA, HENRY D., M.D., Associate in Gynecology and Assistant Demonstrator of Obstetrics in the University of Pennsylvania; Assistant Surgeon to the Gynecean Hospital. 1734 Spruce St.

†1884. BIDDLE, ALEXANDER W., M.D. Chestnut Hill.

1884. BIDDLE, THOMAS, M.D. 122 S. Twenty-second St.

*1903. BIGGS, MONTGOMERY H., M.D. Rutherfordton, N. C.

*1866. BLACK, JOHN JANVIER, A.M. (Princeton), M.D. New Castle, Delaware.

1908. BLAND, P. BROOKE, M.D., Instructor and Demonstrator of Gynecology in Jefferson Medical College; Assistant Gynecologist to the Jefferson Medical College Hospital; Assistant Gynecologist to St. Joseph's and the Philadelphia Hospitals. 1840 S. Broad St.

1894. BLISS, ARTHUR AMES, M.D., Laryngologist and Aurist to the German Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 117 S. Twentieth St.

1894. BOCHROCH, MAX H., M.D., Demonstrator of Neurology and Chief Clinical Assistant in the Nervous Department of the Jefferson Medical College Hospital; Neurologist to the Out-patient Department of St. Joseph's Hospital. 937 N. Eighth St.

1896. BOGER, JOHN A., A.M., M.D., Surgeon to St. Mary's Hospital;

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Surgeon to the Stetson Hospital; Surgeon to the Dispensary of the Episcopal Hospital. 2213 N. Broad St.

1891. BOYD, GEORGE M., M.D., Professor of Clinical Obstetrics in the Medico-Chirurgical College; Obstetrician to the Medico-Chirurgical Hospital; Physician to the Philadelphia Lying-in Charity; Obstetrician and Gynecologist to the Philadelphia Hospital. 1909 Spruce St.

1907. BOYER, HENRY PERCIVAL, M.D., Physician to the Stetson Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

†1884. BRADFORD, T. HEWSON, M.D., Medical Director of the Philadelphia Life Insurance Company; Medical Director of the United Security Life Insurance and Trust Company; Attending Physician to St. Martin's College for Destitute Boys. 125 S. Eighteenth St.

1907. BRADLEY, WILLIAM N., Ph.G., M.D., Assistant Physician to the Medical Dispensary of the Children's Hospital. 1532 S. Sixth St.

1903. BRANSON, THOMAS F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1891. BRINTON, LEWIS, M.D., Chief Physician to the American Hospital for Diseases of the Stomach. 1933 Spruce St.

1900. BRINTON, WARD, M.D., Demonstrator of Physical Diagnosis in the Jefferson Medical College; Assistant in the Medical Dispensary of Jefferson Medical College Hospital. 1423 Spruce St.

1907. BROOKS, MACY, A.B. (Princeton), M.D., Assistant Genito-urinary Surgeon to the Philadelphia Hospital. 1314 Spruce St.

1887. BRUBAKER, ALBERT P., M.D., Professor of Physiology in the Pennsylvania College of Dental Surgery; Adjunct Professor of Physiology and Hygiene in the Jefferson Medical College; Lecturer on Anatomy and Physiology in the Drexel Institute. 105 N. Thirty-fourth St.

*1851. BULLOCK, WILLIAM R., M.D. Tenth and Washington Sts., Wilmington, Del.

1906. BURNS, STILLWELL C., M.D., Lecturer on Surgery in the Medico-Chirurgical College; Assistant Surgeon to the Medico-Chirurgical Hospital. 247 S. Thirteenth St.

1892. BURR, CHARLES W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia Hospital. 1327 Spruce St.

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1906. BUTLER, RALPH, M.D., Chief of the Dispensary for Diseases of the Ear at the University Hospital; Chief of the Dispensary for Diseases of the Ear, Nose, and Throat at the German Hospital; Assistant Laryngologist to the Philadelphia Hospital; Demonstrator of Otology in the Philadelphia Polyclinic. 1824 Chestnut St.

1908. CADBURY, WILLIAM W., A.M., M.D., Pathologist and Member of the Medical Staff of the Henry Phipps Institute; Visiting Physician to the free Hospital for Poor Consumptives, White Haven, Pa.; Examining Physician to the Children's Bureau; Physician to the Starr Centre. 423 S. Fifteenth St.

†1907. CADWALADER, WILLIAMS B., M.D., Pathologist and Clinical Assistant to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Instructor in Nervous Diseases in the Philadelphia Polyclinic. 1710 Locust St.

1905. CAMERON, GEORGE A., M.D., Physician to the Germantown Hospital. S. E. cor. School House Lane and Greene St., Germantown.

1905. CARMANY, HARRY S., M.D., Surgeon to St. Timothy's Hospital, Roxborough; Surgeon to the Dispensary of the Episcopal Hospital. 366 Green Lane, Roxborough.

1905. CARPENTER, HERBERT B., M.D., Physician to the Dispensary of the Children's Hospital. 1805 Spruce St.

1895. CARPENTER, JOHN T., M.D., Lecturer on Ophthalmology in the University of Pennsylvania; Assistant Ophthalmic Surgeon to the University Hospital; Attending Ophthalmologist to the Bryn Mawr Hospital. 1624 Walnut St.

1892. CATTELL, HENRY W., A.M., M.D., Author of *Postmortem Pathology;* American Editor and Translator of Ziegler's *Special Pathological Anatomy;* Editor of Lippincott's *Medical Dictionary.* 3709 Spruce St.

1900. CHANCE, BURTON, M.D., Assistant Surgeon to the Wills Eye Hospital; Ophthalmologist to the Germantown Dispensary and Hospital. 235 S. Thirteenth St.

1885. CHAPIN, JOHN B., M.D., LL.D. (Jefferson and Williams), Physician-in-Chief to the Pennsylvania Hospital for the Insane. Pennsylvania Hospital for the Insane.

1880. CHAPMAN, HENRY C., A.M., M.D., Sc.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in the Jefferson Medical College. 2047 Walnut St.

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1900. CHASE, ROBERT HOWLAND, A.M., M.D., Superintendent of the Friends' Asylum for the Insane. Friends' Asylum, Frankford.

*1868. CHESTON, D. MURRAY, M.D. Harwood P. O., Md.

1897. CHESTON, RADCLIFFE, M.D., Visiting Physician to the Chestnut Hill Hospital; Consulting Physician to the Germantown Hospital and to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill.

1904. CHRISTIAN, HILARY M., M.D., Clinical Professor of Genito-Urinary Diseases in the Medico-Chirurgical College; Professor of Genito-Urinary Diseases in the Philadelphia Polyclinic. 1321 Spruce St.

1903. CHRYSTIE, WALTER, M.D. Bryn Mawr, Pa.

1899. CLARK, JOHN G., M.D., Professor of Gynecology in the University of Pennsylvania; Gynecologist-in-Chief to the University Hospital. 2017 Walnut St.

1897. CLAXTON, CHARLES, A.M., M.D. 5137 Morris St., Germantown.

1872. CLEEMANN, RICHARD A., M.D. 2135 Spruce St.

1896. CLEVELAND, ARTHUR H., M.D., Clinical Professor of Laryngology in the Medico-Chirurgical College; Laryngologist to the Medico-Chirurgical Hospital; Laryngologist and Aurist to the Presbyterian Hospital, and to the Pennsylvania Institution for the Deaf and Dumb. 256 S. Fifteenth St.

1903. COATES, GEORGE MORRISON, A.B., M.D., Professor of Diseases of the Ear in the Philadelphia Polyclinic; Laryngologist to the Henry Phipps Institute for Tuberculosis; Laryngologist to the Pennsylvania Institution for the Instruction of the Blind. 334 S. Nineteenth St.

1908. CODMAN, CHARLES A. E., M.D., Physician to the American Oncologic Hospital. 4116 Spruce St.

1907. COHEN, MYER SOLIS, A.B., M.D., Visiting Physician to the Hospital for Diseases of the Lungs, Chestnut Hill; Assistant Physician to the Philadelphia Hospital; Pediatrician to the Dispensary of the Jewish Hospital; Assistant Physician to the Medical Dispensary of the University Hospital. 4110 Parkside Ave.

1888. COHEN, SOLOMON SOLIS, M.D., Professor of Clinical Medicine in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital, to the Philadelphia Hospital, to the Jewish Hospital, and to the Rush Hospital. 1525 Walnut St.

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1898. COLES, STRICKER, M.D., Assistant Professor of Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson and to the Philadelphia Hospitals. 2103 Walnut St.

1901. COLEY, THOMAS LUTHER, A.B., M.D., Attending Physician to the Methodist Episcopal Hospital of Philadelphia. 257 S. Twenty-first St.

1903. COOPER, J. CARDEEN, M.D., Consulting Physician to the Methodist Home for the Aged. 1016 Lehigh Ave.

1903. COPLIN, W. M. L., M.D., Professor of Pathology in the Jefferson Medical College; Pathologist to and Director of the Laboratories of the Jefferson Medical College Hospital; Pathologist to the Philadelphia Hospital and to the Friends' Asylum for the Insane, Frankford; Bacteriologist to the Pennsylvania State Board of Health. 1529 S. Broad St.

1907. COUNCIL, MALCOLM S., M.D., Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Cathcart Home at Devon. Bryn Mawr, Pa.

1904. CRAIG, FRANK A., M.D., Assistant Instructor in Medicine in the University of Pennsylvania; Member of Staff of the Henry Phipps Institute; Examining Physician to the Free Hospital for Poor Consumptives. 732 Pine St.

1907. CRAMPTON, GEORGE S., M.D. 251 S. Seventeenth St.

1895. CROSS, WILLIAM A., M.D., Consulting Physician to the Jewish Hospital. Jenkintown, Pa.

1904. CRUICE, JOHN M., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Philadelphia Hospital; Physician to the Medical Dispensary of the University Hospital and of St. Joseph's Hospital. 1815 Spruce St.

1902. CURRIE, CHARLES A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1884. CURTIN, ROLAND GIDEON, A.M., M.D., Ph.D., Emeritus Visiting Physician to the Philadelphia Hospital; Consulting Physician to the Presbyterian Hospital; Ex-President of the American Climatological Association; Ex-President of the American Society of Tropical Medicine. 22 S. Eighteenth St.

1884. DA COSTA, JOHN C., M.D., Emeritus Gynecologist to the Jefferson Medical College Hospital; Consulting Gynecologist to St. Agnes's Hospital. 1022 Spruce St.

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1903. Da Costa, John C., Jr., M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College; Chief of the Medical Clinic in the Jefferson Medical College Hospital; Assistant Physician to the Philadelphia Hospital; Hematologist to the German Hospital. 1022 Spruce St.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia and to St. Joseph's Hospitals. 2045 Walnut St.

1887. Daland, Judson, M.D., Professor of Clinical Medicine in the Medico-Chirurgical College; Physician to the Medico-Chirurgical Hospital; Consulting Physician to the Kensington Hospital for Women; Consulting Physician to the State Hospital for the Insane at Norristown. 317 S. Eighteenth St.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital. 5923 Greene St., Germantown.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes's Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1888. Davis, Edward P., A.M., M.D., Professor of Obstetrics in the Jefferson Medical College and in the Philadelphia Polyclinic; Visiting Obstetrician to the Jefferson and to the Polyclinic Hospitals; Obstetrician and Gynecologist to the Philadelphia Hospital; Member of the American Gynecological Society, the American Pediatric Society, and of the International Congress of Obstetrics and Gynecology. 250 S. Twenty-first St.

1889. Davis, Gwilym G., M.D. (Univ. of Penna. and Göttingen), M.R.C.S. England, Associate Professor of Applied Anatomy in the University of Pennsylvania; Surgeon to the Episcopal, to St. Joseph's, and to the Orthopædic Hospitals. 1814 Spruce St.

1900. Davisson, Alex. Heron, M.D. 31 St. Paul's Road, Ardmore.

1894. Deaver, Harry C., M.D., Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Episcopal and to the Stetson Hospitals, to St. Christopher's Hospital for Children, and to the Children's Hospital of the Mary J. Drexel Home. 1534 N. Fifteenth St.

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ELECTED

1887. Deaver, John B., M.D., Surgeon-in-Chief to the German Hospital; Consulting Surgeon to the Germantown Hospital. 1634 Walnut St.

1892. Deaver, Richard Wilmot, M.D., Consulting Surgeon to the Germantown Hospital. 6105 Main St., Germantown.

1902. Dehoney, Howard, M.D. 237 S. Thirteenth St.

1885. Dercum, Francis X., M.D., Professor of Nervous and Mental Diseases in the Jefferson Medical College; Neurologist to the Philadelphia Hospital; Consulting Neurologist to St. Agnes's and to the Jewish Hospitals, and to the State Asylum for the Chronic Insane of Pennsylvania. 1719 Walnut St.

1908. Despard, Duncan L., M.D., Instructor in Surgery in Jefferson Medical College; Chief Assistant in the Surgical Clinic at Jefferson Medical College Hospital. 1806 Pine St.

1908. Dillard, Henry K., Jr., M.D., Physician to the Mary J. Drexel Home at the German Hospital. 234 S. Twentieth St.

1891. Dixon, Samuel G., M.D., President and Executive Curator of the Academy of Natural Sciences of Philadelphia; Member of the Council of the American Philosophical Society; Member of the Board of Trustees of the Wistar Institute of Anatomy; Member of the Board of Managers of the Philadelphia Zoölogical Society. Ardmore, Pa.

*1896. Donnellan, P. S., M.D., L.R.C.S. and P., Ireland. Coronado Beach, Cal.

1897. Dorland, W. A. Newman, M.D., Associate in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Instructor in Obstetrics in the University of Pennsylvania. 1623 Walnut St.

1907. Dorrance, George Morris, M.D., Assistant Surgeon to St. Agnes's Hospital; Assistant Oral Surgeon to the Philadelphia Hospital; Instructor in Surgery in the Philadelphia Polyclinic; Demonstrator of Anatomy in the Dental Department of the University of Pennsylvania. 1716 Locust St.

1902. Dougherty, Sherborne W., M.D., Instructor in Physical Diagnosis in the University of Pennsylvania; Physician to the Dispensary of the German Hospital; Assistant Physician to the Methodist Hospital. 256 S. Sixteenth St.

1893. Downs, Norton, M.D. Fordhooke Farms, Three Tuns, Pa.

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ELECTED

1864. DOWNS, R. N., M.D., Consulting Physician to the Germantown Hospital. 5916 Greene St., Germantown.

1902. DOWNS, ROBERT N., JR., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

1864. DUER, EDWARD L., A.M., M.D., Gynecologist to the Presbyterian Hospital; Consulting Obstetrician to the Maternity Hospital and to the Preston Retreat. 1606 Locust St.

1897. DUER, S. NAUDAIN, M.D., Physician to the Dispensary of the Presbyterian Hospital. 1916 Pine St.

1871. DUHRING, L. A., M.D., Professor of Diseases of the Skin in the University of Pennsylvania; Honorary Member of the Dermatological Society of London, Società Itäliana di Dermatologia e Sifilographia, and Wiener Dermatologische Gesellschaft. 3322 Walnut St.

1881. DULLES, CHARLES WINSLOW, M.D., Lecturer on the History of Medicine in the University of Pennsylvania; Consulting Surgeon of the Rush Hospital. 4101 Walnut St.

*1860. DUNTON, WILLIAM R., M.D. Montrose, Pa.

1899. EDSALL, DAVID L., M.D., Professor of Therapeutics and Pharmacology in the University of Pennsylvania; Physician to the Episcopal Hospital. 1432 Pine St.

*1887. EDWARDS, WILLIAM A., M.D., Professor of Pediatrics in the Medical Department of the University of Southern California. Fourth and Spring Sts., Los Angeles, Cal.

1904. ELMER, WALTER G., M.D., Surgeon to the Out-patient Department of the Presbyterian Hospital; Instructor in Orthopedic Surgery in the University of Pennsylvania; Assistant Orthopedic Surgeon to the University Hospital; Consulting Physician to the Church Home for Children. 1801 Pine St.

1896. ELY, THOMAS C., A.M., M.D. 2041 Green St.

1901. ERCK, THEODORE A., M.D., Assistant Surgeon to the Gynecean Hospital; Associate in Gynecology in the Philadelphia Polyclinic and College for Graduates in Medicine; Gynecologist to the Frederick Douglass Memorial Hospital. 251 S. Thirteenth St.

1893. ESHNER, AUGUSTUS A., M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine; Physician to the Philadelphia Hospital; Assistant Physician to the Orthopædic Hospital and Infirmary for

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ELECTED

Nervous Diseases; Physician to the Hospital for Diseases of the Lungs, Chestnut Hill. 1019 Spruce St.

1905. Evans, Joseph S., Jr., A.B., M.D., Instructor in Clinical Medicine in the University of Pennsylvania; Associate in the William Pepper Laboratory of Clinical Medicine; Assistant Physician to the University Hospital. 2018 Locust St.

1905. Evans, William, M.D. 4009 Chestnut St.

1894. Faries, Randolph, M.D. 2007 Walnut St.

†1903. Farr, Clifford B., A.B., M.D., Professor of Diseases of the Stomach, etc., in the Philadelphia Polyclinic; Instructor in Medicine in the University of Pennsylvania; Physician to the Medical Dispensary of the Hospital of the University of Pennsylvania; Visiting Physician to the Baptist Home. 211 S. Seventeenth St.

1893. Farr, William W., M.D., Physician to the Leamy Home. 117 Allen's Lane, Mount Airy.

1884. Fenton, Thomas H., M.D., Ophthalmologist to St. Vincent's Home, to the Home for Aged Couples, to the Baptist Home, and to the House of the Good Shepherd. 1319 Spruce St.

1907. Ferguson, Albert D., M.D., Visiting Physician to the Widener Memorial School for Crippled Children. 1549 N. Thirteenth St.

1907. Fetterolf, George, A.B., M.D., Laryngologist to the Henry Phipps Institute for Tuberculosis; Laryngologist to the White Haven Sanatorium; Consulting Laryngologist to the Phœnixville Hospital; Demonstrator of Anatomy in the University of Pennsylvania. 330 S. Sixteenth St.

1907. Fife, Charles A., A.B., M.D., Instructor in Diseases of Children, and Associate in the William Pepper Clinical Laboratory in the University of Pennsylvania; Physician to St. Christopher's Hospital for Children, to the Children's Department of the Presbyterian Hospital, and to the Children's Department of the Mary J. Drexel Home; Assistant Pediatrist to the Philadelphia Hospital; Visiting Physician to St. Vincent's Home. 2033 Locust St.

1884. Fisher, Henry M., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital. 1027 Pine St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

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ELECTED

1908. FOULKROD, COLLIN, M.D., Assistant Obstetrician to the Presbyterian Hospital Maternity; Assistant Demonstrator of Obstetrics in Jefferson Medical College; Gynecologist to the Dispensary of the Presbyterian Hospital. 4022 Chestnut St.

1908. FOX, HERBERT, M.D., Chief of the Laboratories of the Pennsylvania Department of Health; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital. 4443 Spruce St.

*†1885. FOX, JOSEPH M., M.D. Leesburg, Va.

1906. FRALEY, FREDERICK, JR., A.B., M.D. 319 S. Seventeenth St.

1903. FRANCINE, ALBERT P., A.M., M.D., Instructor in Medicine in the University of Pennsylvania; Physician to the Medical Dispensary of the University Hospital; Attending Physician to the Philadelphia Hospital, Department of Tuberculosis; Chief Physician to the State Dispensary for Tuberculosis, Philadelphia. 218 S. Fifteenth St.

1897. FRAZIER, CHARLES H., M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University, to the Philadelphia, and to the Episcopal Hospitals; Surgeon to the Home for Crippled Children. 1724 Spruce St.

†1890. FREEMAN, WALTER J., M.D., Emeritus Professor of Laryngology in the Philadelphia Polyclinic; Laryngologist to the Orthopædic Hospital; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 1832 Spruce St.

1893. FRIEBIS, GEORGE, M.D., Ophthalmic Surgeon to the Lutheran Home and Orphanage, Mt. Airy. 1906 Chestnut St.

1889. FUSSELL, M. HOWARD, M.D., Assistant Professor of Medicine in the University of Pennsylvania; Physician to St. Timothy's Hospital; Physician to St. Mary's Hospital. 189 Green Lane, Manayunk.

1899. GAMBLE, ROBERT G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1873. GERHARD, GEORGE S., M.D., Physician-in-Chief to the Bryn Mawr Hospital; Consulting Physician to Bryn Mawr College; Consulting Physician to Villa Nova College. Fifty-eighth Street and Overbrook Ave.

1902. GHRISKEY, ALBERT A., M.D. 3936 Walnut St.

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ELECTED

1892. GIBB, JOSEPH S., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Surgeon to the Ear, Nose, and Throat Department of the Episcopal Hospital. 1907 Chestnut St.

1899. GIBBON, JOHN H., M.D., Professor of Surgery in the Jefferson Medical College; Surgeon to the Pennsylvania and to the Bryn Mawr Hospitals; Consulting Surgeon to the Woman's Hospital. 1608 Spruce St.

1908. GILDERSLEEVE, NATHANIEL, M.D. Dormitories, University of Pennsylvania.

1897. GIRVIN, JOHN H., M.D., Physician for Diseases of Women at the Presbyterian Hospital; Instructor in Obstetrics in the University of Pennsylvania. 3924 Walnut St.

1889. GITHENS, WILLIAM H. H., M.D., Visiting Physician to the Sheltering Arms. 1337 Pine St.

1906. GITTINGS, J. CLAXTON, M.D., Visiting Physician to the Children's Hospital of the Mary J. Drexel Home, and to the Medical Dispensary of the Children's and the Presbyterian Hospitals; Assistant Pediatric Physician to the University Hospital; Instructor in Pediatrics in the University of Pennsylvania. 3942 Chestnut St.

1905. GIVEN, ELLIS E. W., M.D., Surgeon to the William L. Elkins Masonic Orphanage for Girls; Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to the Dispensary of St. Christopher's Hospital for Children. 2714 Columbia Ave.

1894. GLEASON, E. B., M.D., Clinical Professor of Otology in the Medico-Chirurgical College; Surgeon-in-Charge of the Nose, Throat, and Ear Department of the Northern Dispensary. 2033 Chestnut St.

1884. GODEY, HARRY, M.D. Aldine Hotel.

1906. GOEPP, R. MAX, M.D., Professor of Clinical Medicine and Dean of the Philadelphia Polyclinic and College for Graduates in Medicine. 332 S. Fifteenth St.

1906. GOLDBERG, HAROLD G., M.D., Curator and Pathologist to the Wills Eye Hospital; First Lieutenant and Assistant Surgeon to the Second Troop, Philadelphia City Cavalry, N. G. P. 1905 Chestnut St.

1893. GOODELL, W. CONSTANTINE, M.D. 300 S. Thirteenth St.

1908. GOODMAN, EDWARD H., M.D., Assistant Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Medical Dispensary of the University

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ELECTED

Hospital; Dispensary Physician to the Presbyterian Hospital. 2035 Chestnut St.

1905. GORDON, ALFRED, M.D., Associate in Nervous and Mental Diseases in the Jefferson Medical College; Examiner of the Insane at the Philadelphia Hospital. 1430 Pine St.

*†1897. GOULD, GEORGE M., A.M., M.D. The Sentinels, Cayuga Heights, Ithaca, N. Y.

1894. GRAHAM, EDWIN E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and to the Philadelphia Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1885. GRAHAM, JOHN, M.D. 326 S. Fifteenth St.

1904. GRAYSON, CHARLES P., M.D., Clinical Professor of Laryngology and Rhinology in the University of Pennsylvania; Physician-in-Charge of Throat and Nose Department of the University Hospital; Otolaryngologist to the Philadelphia Hospital. 251 S. Sixteenth St.

1883. GRIFFITH, J. P. CROZER, M.D., Clinical Professor of the Diseases of Children in the University of Pennsylvania; Physician to St. Agnes's, to the Children's, and to the Methodist Hospitals. 1810 Spruce St.

1902. GWYN, NORMAN B., M.B., Instructor in Medicine in the University of Pennsylvania. 23 S. Twenty-first St.

1894. HAMILL, SAMUEL McC., M.D., Professor of Diseases of Children in the Philadelphia Polyclinic and College for Graduates in Medicine; Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. HAND, ALFRED, JR., M.D., Visiting Physician to the Children's Hospital, to the Children's Hospital of the Mary J. Drexel Home of Philadelphia, and to the Methodist Hospital. 1724 Pine St.

1886. HANSELL, HOWARD F., M.D., Professor of Ophthalmology in the Jefferson Medical College; Ophthalmic Surgeon to the Philadelphia Hospital and to the Jefferson Medical College Hospital; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic. 1528 Walnut St.

1889. HARE, HOBART A., M.D., Professor of Therapeutics in the Jefferson Medical College; Physician to the Jefferson Medical College Hospital. 1801 Spruce St.

1865. HARLAN, GEORGE C., M.D., Consulting Surgeon to the Wills Eye Hospital; Ophthalmologist to the Pennsylvania Hos-

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ELECTED

pital; Consulting Ophthalmologist to the Pennsylvania Institution for the Blind, and to the Pennsylvania Institution for the Deaf. 1700 Walnut St.

1903. HART, CHARLES D., M.A., M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Inspector of the Eastern Penitentiary. 1317 Walnut St.

1885. HARTE, RICHARD H., M.D., Adjunct Professor of Surgery in the University of Pennsylvania; Surgeon to the Pennsylvania and to the Orthopædic Hospitals; Consulting Surgeon to St. Mary's, to St. Timothy's, and to the Bryn Mawr Hospitals. 1503 Spruce St.

1888. HARTZELL, MILTON B., M.D., Associate in Dermatology in the University of Pennsylvania; Clinical Professor of Dermatology in the Woman's Medical College of Pennsylvania; Dermatologist to the Philadelphia Hospital. 3644 Chestnut St.

1907. HATFIELD, CHARLES JAMES, A.B. (Princeton), M.D., Physician to the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. 2008 Walnut St.

1872. HAYS, I. MINIS, M.D. 266 S. Twenty-first St.

1882. HEARN, W. JOSEPH, M.D., Professor of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia Hospital. 1120 Walnut St.

1908. HEINEBERG, ALFRED, M.D., Demonstrator of Gynecology in the Jefferson Medical College; Assistant Gynecologist to St. Agnes's Hospital; Instructor in Therapeutics and Matera Medica in the Jefferson Medical College, and in the Philadelphia College of Pharmacy. 1327 Pine St.

1901. HEISLER, JOHN C., M.D., Professor of Anatomy in the Medico-Chirurgical College. 3829 Walnut St.

1884. HENRY, FREDERICK P., M.D., Professor of the Principles and Practice of Medicine in the Woman's Medical College of Pennsylvania; Physician to the Philadelphia Hospital; Corresponding Member of the Royal Academy of Medicine of Rome. 114 S. Eighteenth St.

1903. HENRY, J. NORMAN, M.D., Physician to the Out-patient Department of the Pennsylvania Hospital; Assistant Physician to the Philadelphia Hospital; Clinical Instructor in Medicine in the Woman's Medical College of Pennsylvania; Physician-in-Charge of the De Lancey School. 114 S. Eighteenth St.

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ELECTED

1891. HEWSON, ADDINELL, A.M., M.D., Professor of Anatomy in the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to St. Timothy's Hospital, Roxborough. 2120 Spruce St.

1872. HINKLE, A. G. B., M.D. 1300 Spring Garden St.

1897. HINKLE, WILLIAM M., M.D., Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 1323 N. Thirteenth St.

*1892. HINSDALE, GUY, A.M., M.D., Secretary of the American Climatological Association; Corresponding Member of the British Balneological and Climatological Society; Fellow of the American Academy of Medicine; Lecturer on Climatology in the Medico-Chirurgical College, Philadelphia. Hot Springs, Va.

1888. HIRSH, A. BERN, M.D., Physician to the Home for Aged Couples. 1711 Diamond St.

1888. HIRST, BARTON COOKE, M.D., Professor of Obstetrics in the University of Pennsylvania; Gynecologist to the Philadelphia and to the Howard Hospitals. 1821 Spruce St.

1903. HIRST, JOHN COOKE, M.D., Assistant Obstetrician to the University and the Philadelphia Hospitals; Attending Physician to the Maternity Hospital and to the Gynecological Dispensary of the Howard Hospital; Consulting Obstetrician to the Southeastern Dispensary. 1618 Pine St.

1908. HITCHENS, ARTHUR PARKER, M.D., Special Lecturer in the Medico-Chirurgical College. Glenolden, Pa.

*1894. HOCH, WILLIAM R., M.D. 108 Central Block, Pueblo, Colorado.

1905. HODGE, EDWARD B., JR., M.D., Surgeon to the Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Orthopædic Hospital; Dispensary Surgeon to the Presbyterian Hospital; Surgeon to the Children's Hospital. 346 S. Sixteenth St.

1885. HOLLAND, JAMES W., M.D., Professor of Medical Chemistry and Toxicology in the Jefferson Medical College. 2006 Chestnut St.

1906. HOLLOWAY, THOMAS B., M.D., Assistant Surgeon to the Dispensary for Diseases of the Eye at the University Hospital; Assistant Ophthalmologist to the Orthopædic Hospital; Registrar of the Ophthalmic Wards of the Philadelphia Hospital; Ophthalmologist to the Philadelphia Orphanage. 1819 Chestnut St.

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ELECTED

1888. HORWITZ, ORVILLE, M.D., Professor of Genito-urinary Surgery in the Jefferson Medical College; Surgeon to the Jefferson Medical College Hospital, to St. Agnes's Hospital, and to the State Hospital for the Insane; Consulting Surgeon to the Jewish Hospital. 1721 Walnut St.

1908. HOYT, DANIEL M., M.D., Assistant Demonstrator of Pharmacology in the University of Pennsylvania; Physician of the Medical Dispensary of the University Hospital. 3604 Chestnut St.

1892. HUGHES, WILLIAM E., M.D., Visiting Physician to the Philadelphia Hospital; Pathologist to the Presbyterian Hospital. 3945 Chestnut St.

1898. HUTCHINSON, JAMES P., M.D., Surgeon to the Pennsylvania, to the Methodist, to the Children's, to St. Timothy's, and to the Bryn Mawr Hospitals; Adjunct Professor of Surgery in the University of Pennsylvania. 1702 Locust St.

1871. INGHAM, JAMES V., M.D. 1811 Walnut St.

*1885. JACKSON, EDWARD, M.D., Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Ophthalmologist to the Denver City and County Hospital. 1434 Glenarm St., Denver, Col.

1906. JACOBS, FRANCIS BRINTON, B.S., M.D., Pediatrist to the Howard Hospital; Pediatrist to the American Hospital for Diseases of the Stomach; Visiting Physician to the Philadelphia Orphan Asylum; Assistant in the Laboratory of the Polyclinic Hospital. 334 S. Nineteenth St.

1887. JAYNE, HORACE, M.D., Ph.D. 318 S. Nineteenth St.

1898. JOHNSON, RUSSELL H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb; Physician to the Chestnut Hill Hospital. Chestnut Hill, Philadelphia.

1900. JONES, CHARLES JAMES, A.M., M.D., Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Assistant Surgeon to the Wills Eye Hospital. 1009 N. Sixth St.

1900. JOPSON, JOHN H., M.D., Surgeon to the Children's, to the Presbyterian, and to the Bryn Mawr Hospitals, and to the Philadelphia Home for Incurables. 1824 Pine St.

1900. JUDSON, CHARLES F., A.B., M.D., Physician to St. Christopher's Hospital for Children, to the Southern Home for

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ELECTED

Destitute Children, and to the Sheltering Arms. 1539 Pine St.

1902. JUMP, HENRY D., M.D., Instructor in Medicine in the University of Pennsylvania; Assistant Physician to the Medical Dispensary of the University Hospital. 4634 Chester Ave.

1886. JURIST, LOUIS, M.D. 916 N. Broad St.

1903. KALTEYER, FREDERICK J., M.D., Demonstrator of Clinical Medicine in the Jefferson Medical College; Chief of the Out-patient Department, Assistant Attending Physician and Hematologist to the Jefferson Medical College Hospital; Pathologist to the Philadelphia Lying-in-Charity. 1533 Pine St.

1904. KANE, J. A. BAYARD, M.D. 1529 Spruce St.

†1867. KEEN, WILLIAM W., M.D., LL.D. (Hon.), F.R.C.S. (Eng. and Edin.), Emeritus Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Membre Correspondant Étranger de la Société de Chirurgie de Paris; Honorary Member of the Société Belge de Chirurgie and of the Clinical Society of London; Ehrenmitglied der deutschen Gesellschaft für Chirurgie. 1729 Chestnut St.

1897. KELLY, ALOYSIUS O. J., A.M., M.D., Assistant Professor of Medicine in the University of Pennsylvania; Professor of the Theory and Practice of Medicine in the University of Vermont; Professor of Pathology in the Woman's Medical College of Pennsylvania; Physician to St. Agnes's Hospital, and Pathologist to the German Hospital. 1911 Pine St.

*1887. KELLY, HOWARD A., M.D., Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md. 1406 Eutaw Place, Baltimore, Md.

1898. KEMPTON, AUGUSTUS F., M.D. 2118 Pine St.

1905. KERCHER, DELNO E., M.D. 1927 S. Eighteenth St.

1902. KING, WILLIAM HOWARD, M.D. 103 Narberth Ave., Narberth, Pa.

*1907. KINYOUN, JOSEPH J., M.D., Ph.D. (Georgetown), Professor of Pathology and Bacteriology in the George Washington University. 1423 Clifton St., Washington, D. C.

1895. KNEASS, SAMUEL S., M.D., Associate in the William Pepper Laboratory of Clinical Medicine in the University of Pennsylvania. 1510 Walnut St.

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ELECTED

1908. KNIPE, JAY C., M.D., Ophthalmologist to the Jewish Hospital; Assistant Ophthalmologist to the Philadelphia Hospital, and to the Mary J. Drexel Home; Chief of the Eye Clinic at the Jefferson Medical College Hospital; Demonstrator of Osteology and Syndesmology in the Jefferson Medical College. 2035 Chestnut St.

1808. KNOWLES, FRANK CROZER, M.D., Assistant Dermatologist to the Philadelphia Hospital; Dermatologist to the Northern Dispensary, the Church Home for Children, and the St. Vincent's Home; Assistant Dermatologist to the Hospital of the Woman's Medical College, and to the Dispensary of the Children's Hospital; Associate Dermatologist to the Dispensary of the Howard Hospital. 332 South Seventeenth St.

1904. KRAUSS, FREDERICK, M.D., Ophthalmic Surgeon to St. Christopher's Hospital for Children and Dispensary; Instructor in Ophthalmology in the Philadelphia Polyclinic. 1701 Chestnut St.

1905. KREMER, WALTER H., M.D. 6122 Main St., Germantown.

1900. KRUSEN, WILMER, M.D., Instructor in Gynecology in Jefferson Medical College; Assistant Gynecologist and Chief of the Gynecological Dispensary of St. Joseph's Hospital; Consulting Gynecologist to the Children's Hospital in Germantown. 127 N. Twentieth St.

1897. KYLE, D. BRADEN, A.M., M.D., Professor of Laryngology in the Jefferson Medical College; Consulting Laryngologist, Rhinologist, and Otologist to St. Agnes's Hospital and to the Philadelphia Home for Incurables; Laryngologist to the New Jersey Training School for Feeble-minded Children; Bacteriologist to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1517 Walnut St.

1904. LANDIS, HENRY R. M., M.D., Visiting Physician to the White Haven Sanatorium; Member of the Staff of the Henry Phipps Institute; Assistant Visiting Physician to the Philadelphia Hospital. 11 S. Twenty-first St.

1907. LANGDON, H. MAXWELL, M.D., Instructor in Ophthalmology in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Diseases of the Eye in the University Hospital; Assistant Ophthalmologist to the Orthopædic Hospital; Ophthalmologist to the Methodist Episcopal Home for the Aged. 1728 Chestnut St.

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ELECTED

1908. LAVENSON, RALPH S., M.D., Assistant Physician to the Philadelphia Hospital; Assistant Pathologist to the University Hospital; Associate in the William Pepper Laboratory of Clinical Medicine; Assistant Demonstrator of Gross Morbid Anatomy in the University of Pennsylvania. 1218 Locust St.

1904. LE BOUTILLIER, THEODORE, M.D., Clinical Professor of Pediatrics in the Woman's Medical College of Pennsylvania; Physician to the Dispensary of St. Christopher's Hospital for Children; Pediatrist to the Woman's College Hospital Dispensary; Visiting Physician to the Baptist Orphanage and to the Southern Home for Destitute Children. 216 S. Twentieth St.

1893. LE CONTE, ROBERT G., A.B., M.D., Surgeon to the Pennsylvania and the Bryn Mawr Hospitals; Consulting Surgeon to the Germantown and the Gynecean Hospitals. 1530 Locust St.

1887. LEAMAN, HENRY, M.D. 832 N. Broad St.

1908. LEE, WALTER E., M.D., Chief Resident Physician to the Pennsylvania Hospital. Pennsylvania Hospital.

1903. LEFFMANN, HENRY, A.M., D.D.S., Ph.D., Professor of Chemistry in the Woman's Medical College of Pennsylvania; Professor of Chemistry in the Wagner Free Institute of Science; Emeritus Professor of Chemistry in the Pennsylvania College of Dental Surgery; Pathological Chemist to the Jefferson Medical College Hospital. 1839 N. Seventeenth St.

1892. LEIDY, JOSEPH, M.D., Officier l'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1319 Locust St.

1903. LEONARD, CHARLES LESTER, M.D. 112 S. Twentieth St.

1877. LEWIS, MORRIS J., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases and to the Pennsylvania Hospital. 1316 Locust St.

1904. LINDAUER, EUGENE, M.D., Instructor of Neurology in the Medico-Chirurgical Hospital; Associate in Clinical Medicine in the Philadelphia Polyclinic; Assistant Neurologist to the Philadelphia Hospital. 2018 N. Thirty-second St.

1886. LLOYD, J. HENDRIE, A.M. (Princeton), M.D., Neurologist to the Philadelphia Hospital; Physician to the Methodist Episcopal Hospital, and to the Home for Crippled Children;

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ELECTED

Consulting Neurologist to the State Asylum for the Chronic Insane of Pennsylvania and to the Pennsylvania Training School for Feeble-minded Children. 3918 Walnut St.

1900. LODGE, JOHN W., M.D., Consulting Physician to the Bryn Mawr Hospital. Merion, Pa.

1907. LODHOLZ, EDWARD, M.D., Demonstrator of Physiology in the University of Pennsylvania. 3103 Diamond St.

1893. LONGAKER, DANIEL, M.D., Surgeon to the Jewish Maternity Hospital. 1402 N. Sixteenth St.

1906. LONGCOPE, WARFIELD T., M.D., Director of the Ayer Clinical Laboratory of the Pennsylvania Hospital; Instructor in Medicine in the University of Pennsylvania. 323 S. Sixteenth St.

1877. LONGSTRETH, MORRIS, M.D. 1416 Spruce St.

1907. LOUX, HIRAM R., M.D. 1614 N. Broad St.

1900. MCCARTHY, DANIEL J., M.D., Instructor in Neurology in the Philadelphia Polyclinic; Associate in the William Pepper Laboratory of Clinical Medicine. 1329 Spruce St.

1875. MCCLELLAN, GEORGE, M.D., Professor of Applied Anatomy in the Jefferson Medical College; Consulting Surgeon to the Howard Hospital. 1116 Spruce St.

*1903. MCCONNELL, GUTHRIE, M.D., Bacteriologist to the Missouri State Board of Health; Pathologist to the St. Louis Skin and Cancer Hospital, and to St. Luke's Hospital. 4175 Washington Ave., St. Louis, Mo.

1895. MCFARLAND, JOSEPH, M.D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College; Pathologist to the Medico-Chirurgical and the Philadelphia Hospitals. 442 W. Stafford St., Germantown.

1905. MCKENZIE, ROBERT TAIT, A.B., M.D., Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 26 S. Twenty-first St.

*1900. MCREYNOLDS, ROBERT PHILLIPS, M.D. 213 S. Broadway, Los Angeles, Cal.

1886. MACCOY, ALEXANDER W., M.D., Surgeon for Diseases of the Nose and Throat in the Out-patient Department of the Pennsylvania Hospital; Member of the American Laryngological Society. 216 S. Fifteenth St.

1901. MACLEOD, GEORGE I., JR., M.D., Physician to the Bryn Mawr Hospital; Assistant Physician to Bryn Mawr College. Ardmore, Pa.

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ELECTED

1896. MAKUEN, G. HUDSON, M.D., Professor of Defects of Speech in the Philadelphia Polyclinic; Laryngologist to St. Mary's Hospital and to the Frederick Douglass Memorial Hospital; Visiting Consultant on Defects of Speech to the New Jersey Training School for Feeble-minded Children. 1627 Walnut St.

*1885. MALLET, JOHN WILLIAM, M.D., Ph.D. (Goett.), LL.D. (Princeton and Johns Hopkins), F.R.S., Professor of Chemistry in the University of Virginia. University of Virginia, Charlottesville, Va.

1898. MARSHALL, GEORGE MORLEY, M.D., Laryngologist to the Philadelphia Hospital; Physician and Laryngologist to St. Joseph's Hospital. 1819 Spruce St.

1893. MARSHALL, JOHN, M.D., Nat.Sc.D., LL.D., Professor of Chemistry and Toxicology in the University of Pennsylvania. 1718 Pine St.

1889. MARTIN, EDWARD, M.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to University, Howard, Philadelphia, and Bryn Mawr Hospitals. 1506 Locust St.

1885. MAYS, THOMAS J., M.D., Visiting Physician to the Rush Hospital. 1829 Spruce St.

*1868. MEARS, J. EWING, M.D. 1535 Land Title Building, Broad and Sansom Sts.

1875. MEIGS, ARTHUR V., M.D., Physician to the Pennsylvania Hospital; Consulting Physician to the Pennsylvania Institution for the Instruction of the Blind. 1322 Walnut St.

*1894. MILLER, D. J. MILTON, M.D., Visiting Physician to the Children's Hospital. 1700 Pacific Ave., Atlantic City, N. J.

1881. MILLS, CHARLES K., M.D., Professor of Neurology in the University of Pennsylvania; Neurologist to the Philadelphia Hospital; Consultant to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1909 Chestnut St.

1904. MITCHELL, CHARLES F., M.D., Surgeon to the Germantown Hospital and to the Out-patient Department of the Pennsylvania Hospital; Assistant Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases. 251 S. Seventeenth St.

†1888. MITCHELL, JOHN K., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Assistant Neurologist to the Presbyterian Hospital; Attending

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ELECTED

Physician to the Pennsylvania Training School for Feeble-minded Children. 1730 Spruce St.

1856. MITCHELL, S. WEIR, M.A., M.D., LL.D. (Edinburgh, Harvard, Princeton, and Toronto), Foreign Correspondent (1st Div.) French Academy of Medicine; Corresponding Member of the Verein für Innerer Medicin, Berlin; Honorary Member of the Royal Medico-Chirurgical Society of London; Doctor of Medicine, Honoris Causa, University of Bologna. 1524 Walnut St.

1908. MONTGOMERY, CHARLES M., A.B., M.D., Physician to the Henry Phipps Institute; Physician to the Home for Consumptives at Chestnut Hill; Physician to the Dispensary of the Episcopal Hospital; Examining Physician for the White Haven Sanatorium. 323 S. Sixteenth St.

1882. MONTGOMERY, EDWARD E., M.D., Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Jefferson Medical College Hospital and to St. Joseph's Hospital. 1703 Walnut St.

1886. MORRIS, CASPAR, M.D. 2050 Locust St.

1893. MORRIS, ELLISTON J., M.D., Physician to the Episcopal Hospital and to the Midnight Mission. 128 S. Eighteenth St.

1883. MORRIS, HENRY, M.D., Professor of Anatomy in the Woman's Medical College of Pennsylvania; Senior Visiting Physician to St. Joseph's Hospital; Associate Member, Military Surgeons of U. S. A. 313 S. Sixteenth St.

1856. MORRIS, J. CHESTON, M.D. 1514 Spruce St.

1906. MORRISON, WILLIAM H., M.D., Surgeon to the Frankford Hospital. 8021 Frankford Ave.

1897. MORTON, SAMUEL W., M.D. 1933 Chestnut St.

1891. MORTON, THOMAS S. K., M.D. School House Lane, Germantown.

1904. MOULTON, ALBERT R., M.D., Senior Assistant Physician to the Pennsylvania Hospital for the Insane; Physician to the Out-patient Department for Nervous and Mental Diseases at the Pennsylvania Hospital; Professor of Mental Diseases in the Maine Medical School (Bowdoin College). Pennsylvania Hospital for the Insane.

1905. MÜLLER, GEORGE P., M.D., Instructor in Surgery in the University of Pennsylvania; Assistant Surgeon to the Philadelphia Hospital; Assistant Surgeon to the Home for Crippled Children; Assistant Pathologist to the German Hospital. 314 S. Fifteenth St.

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ELECTED

1882. MUSSER, JOHN H., M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University, the Philadelphia, and the Presbyterian Hospitals. 1927 Chestnut St.

1905. MUTSCHLER, LOUIS H., M.D., Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to the Dispensary of St. Christopher's Hospital for Children; Assistant Surgeon to the Orthopædic Hospital. 2030 Tioga St.

1896. MYERS, T. D., M.D. 1521 Spruce St.

1902. NASSAU, CHARLES F., M.D., Surgeon to St. Joseph's Hospital; Consulting Surgeon to the Frankford Hospital; Assistant Surgeon to the Jefferson Medical College Hospital; Demonstrator of Applied Anatomy in Jefferson Medical College. 1831 Chestnut St.

1886. NEFF, JOSEPH S., M.D. 580 City Hall.

1887. NEILSON, THOMAS RUNDLE, A.M., M.D., Surgeon to the Episcopal Hospital and to St. Christopher's Hospital for Children; Clinical Professor of Genito-urinary Diseases in the University of Pennsylvania. 122 S. Seventeenth St.

1905. NEWCOMET, WILLIAM S., M.D. 3501 Baring St.

1905. NEWLIN, ARTHUR, M.D., Physician to the Dispensary of the Children's Hospital; Assistant Physician to the Orthopædic Hospital. 253 S. Thirteenth St.

1899. NICHOLSON, WILLIAM R., JR., M.D., Assistant Instructor in Obstetrics in the University of Pennsylvania; Obstetrician to the Maternity Hospital; Assistant Surgeon to the Gynecean Hospital. 350 S. Fifteenth St.

1889. NOBLE, CHARLES P., M.D., Surgeon-in-Chief to the Kensington Hospital for Women; Surgeon-in-Charge of the Department for Women of the Northern Dispensary and of the Union Mission Hospital; Clinical Professor of Gynecology in the Woman's Medical College of Pennsylvania; Lecturer on Gynecology in the Philadelphia Polyclinic. 1509 Locust St.

1898. NOLAN, EDWARD J., M.D., Recording Secretary and Librarian of the Academy of Natural Sciences of Philadelphia. 825 N. Twentieth St.

1905. NORRIS, CHARLES C., M.D., Instructor in Gynecology in the University of Pennsylvania; Obstetrician and Assistant Attending Physician to the Maternity Hospital; Gynecologist to the Philadelphia Hospital. 1503 Locust St.

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ELECTED

1905. NORRIS, GEORGE WILLIAM, A.B., M.D., Associate in Medicine in the University of Pennsylvania; Assistant Physician to the Philadelphia Hospital; Physician to the Tuberculosis Dispensary of the Commonwealth of Pennsylvania. 1530 Locust St.

*1901. NORRIS, HENRY, M.D. Rutherfordton, N. C.

*1865. NORRIS, ISAAC, M.D. Care Morgan, Harjes & Co., Paris, France.

1892. NORRIS, RICHARD C., M.D., Lecturer on Clinical and Operative Obstetrics in the University of Pennsylvania; Obstetrician in Charge to the Preston Retreat; Visiting Obstetrician to the Philadelphia Hospital; Gynecologist to the Methodist Episcopal Hospital and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1884. OLIVER, CHARLES A., A.M., M.D., Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to the Philadelphia and to the Presbyterian Hospitals; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania. 1507 Locust St.

*1884. O'NEILL, J. WILKS, M.D. Bergenfield, N. J.

*1885. OSLER, WILLIAM, M.D., Regius Professor of Medicine in Oxford University, England. No. 7, Norman Gardens, Oxford, England.

1903. OSTHEIMER, MAURICE, M.D., Senior Instructor in Pediatrics in the University of Pennsylvania; Physician-in-Charge of the Children's Dispensary, University Hospital; Visiting Physician to the Baptist Orphanage. 225 S. Twentieth St.

1897. PACKARD, FRANCIS R., M.D., Aurist to the Out-patient Department of the Pennsylvania Hospital; Professor of Diseases of the Ear in the Philadelphia Polyclinic and College for Graduates in Medicine; Consulting Aurist to the Bryn Mawr Hospital. 1836 Pine St.

1898. PAGE, HENRY F., M.D., Assistant Physician to the German Hospital and Physician to the Medical Dispensary of the same; Instructor in Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to the Baptist Home. 1907 Girard Ave.

1906. PANCOAST, HENRY K., M.D., Lecturer on Skiagraphy in the University of Pennsylvania; Skiagrapher to the University Hospital. 4238 Pine St.

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ELECTED

1899. PARKE, WILLIAM E., M.D., Consulting Obstetrician to the Episcopal Hospital; Assistant Surgeon in the Kensington Hospital for Women; Associate Surgeon in the Department for Diseases of Women of the Northern Dispensary. 1739 N. Seventeenth St.

*1903. PEARCE, RICHARD M., M.D., Professor of Pathology in the University and Bellevue Hospital Medical College. Carnegie Laboratory, 338 E. Twenty-sixth St., New York.

†1889. PENROSE, CHARLES BINGHAM, M.D., Ph.D. (Harvard), Formerly Professor of Gynecology in the University of Pennsylvania. 1720 Spruce St.

1854. PENROSE, R. A. F., M.D., LL.D., Professor (Emeritus) of Obstetrics and Diseases of Women and Children in the University of Pennsylvania. 1331 Spruce St.

†1902. PEPPER, WILLIAM, M.D., Instructor in Medicine in the University of Pennsylvania. 1811 Spruce St.

1884. PERKINS, FRANCIS M., M.D., Ophthalmic Surgeon to St. Agnes's Hospital. 1428 Pine St.

1905. PFAHLER, GEORGE E., M.D., Adjunct Professor of Symptomatology in the Medico-Chirurgical College; Director of the Röntgen Ray Laboratory, and Assistant Physician to the Medico-Chirurgical Hospital. 1321 Spruce St.

1907. PFROMM, GEORGE W., Ph.G., M.D. 1434 N. Fifteenth St.

1907. PHILLIPS, HORACE, M.D., Second Assistant Physician to the Pennsylvania Hospital for the Insane; Visiting Physician to the Eastern Penitentiary of Pennsylvania. Pennsylvania Hospital for the Insane.

*1899. PHILLIPS, JOHN L., M.D. The Bellefield, Pittsburgh, Pa.

1883. PIERSOL, GEORGE A., M.D., Professor of Anatomy in the University of Pennsylvania. 4722 Chester Ave.

1905. PITFIELD, ROBERT L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1896. POSEY, WILLIAM CAMPBELL, M.D., Surgeon to the Wills Eye Hospital; Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Ophthalmic Surgeon to the Howard Hospital; Ophthalmologist to the Pennsylvania Hospital for Epileptics. 2049 Chestnut St.

1902. POTTS, BARTON H., M.D., Laryngologist and Aurist to the German and to St. Mary's Hospitals; Assistant Aural Surgeon and Chief of the Ear Dispensary of the Children's

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ELECTED

Hospital; Laryngologist to the Pennsylvania Training School for Feeble-minded Children. 109 S. Twentieth St.

1899. POTTS, CHARLES S., M.D., Professor of Neurology in the Medico-Chirurgical College; Neurologist to the Philadelphia Hospital; Consultant to the Insane Department of the Philadelphia Hospital; Consultant to the Hospital for the Insane, Atlantic County, New Jersey. 1728 Chestnut St.

1905. PRENDERGAST, MICHAEL T., M.D. 2435 Columbia Ave.

1907. PRICE, GEORGE E., M.D., Associate in Nervous and Mental Diseases in the Jefferson Medical College; Chief Clinical Assistant in the Neurological Dispensary of the Jefferson Medical College Hospital; Assistant Neurologist to the Philadelphia Hospital. 126 S. Seventeenth St.

†1889. PRICE, JOSEPH, M.D., Obstetric Physician to the Philadelphia Dispensary. 241 N. Eighteenth St.

1903. PURVES, GEORGE MOREHOUSE, M.D., Registrar of the Surgical Dispensary of the Hospital of the University of Pennsylvania. 4204 Walnut St.

†1903. PYLE, WALTER L., A.M., M.D., Member of the American Ophthalmological Society; Assistant Surgeon to the Wills Eye Hospital. 1931 Chestnut St.

1908. RADCLIFFE, MCCLUNEY, A.M. (Lafayette), M.D., Attending Surgeon to the Wills Eye Hospital. 1812 Chestnut St.

1887. RANDALL, B. ALEXANDER, M.A., M.D., Clinical Professor of Diseases of the Ear in the University of Pennsylvania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb, and to St. Timothy's Hospital. 1717 Locust St.

*1904. RAVENEL, MAZYCK P., M.D., Professor of Bacteriology in the University of Wisconsin. Madison, Wis.

1887. REED, CHARLES H., M.D. 121 S. Seventeenth St.

1885. REICHERT, EDWARD T., M.D., Professor of Physiology in the University of Pennsylvania. University of Pennsylvania.

1897. RHEIN, JOHN H. W., M.D., Neurologist to the Howard Hospital; Physician to the Philadelphia Home for Incurables; Bacteriologist to the Pennsylvania Training School for Feeble-minded Children. 1732 Pine St.

1906. RHEIN, ROBERT D., M.D., Assistant Physician to the Howard Hospital; Associate Physician to the American Hospital for Diseases of the Stomach; Examining Phy-

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ELECTED

sician to the Free Hospital for Poor Consumptives. 2016 Pine St.

1891. RHOADS, EDWARD G., M.D. 159 W. Coulter St., Germantown.

1898. RIESMAN, DAVID, M.D., Professor of Clinical Medicine in the Philadelphia Polyclinic; Associate in Medicine in the University of Pennsylvania; Physician to the Philadelphia and to the Jewish Hospitals. 1715 Spruce St.

1895. RING, G. ORAM, A.M., M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. 2014 Chestnut St.

1905. RISLEY, J. NORMAN, M.D., Assistant Surgeon to the Wills Eye Hospital; Ophthalmologist to the Pennsylvania Training School for Feeble-minded Children. 1728 Chestnut St.

1891. RISLEY, S. D., M.D., Attending Surgeon to the Wills Eye Hospital; Professor (Emeritus) of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine; Member of the Board of Managers of the Pennsylvania Training School for Feeble-minded Children; Alumnus Manager of the University Hospital. 1728 Chestnut St.

†1878. ROBERTS, JOHN B., M.D., Professor of Surgery in the Philadelphia Polyclinic; Surgeon to the Methodist Episcopal Hospital. 313 S. Seventeenth St.

1899. ROBERTS, WALTER, M.D., Professor of Otology in the Philadelphia Polyclinic; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia Hospital. 1732 Spruce St.

1903. ROBERTSON, WILLIAM EGBERT, M.D., Assistant Visiting Physician and Chief of Dr. Daland's Clinic in the Medico-Chirurgical Hospital; Lecturer to Nurses in the Medico-Chirurgical Hospital; Physician to the Episcopal Hospital. 320 S. Sixteenth St.

1907. ROBINSON, EDWIN TAYLOR, M.D. 1906 Pine St.

1902. ROBINSON, JAMES WEIR, M.D., Assistant Surgeon to the Presbyterian Hospital. 326 S. Sixteenth St.

1903. ROBINSON, WILLIAM DUFFIELD, Ph.G., M.D. 2012 Mount Vernon St.

1900. RODMAN, WILLIAM L., M.D., Professor of Surgery in the Medico-Chirurgical College; Surgeon to the Presbyterian

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ELECTED

Hospital; Surgeon to the Philadelphia Hospital. 1904 Chestnut St.

1898. Ross, GEORGE G., M.D., Assistant Surgeon to the German Hospital and Surgeon to the Out-patient Department of the same; Surgeon to the Germantown Hospital. 1721 Spruce St.

*1907. ROYER, B. FRANKLIN, M.D. Donaldson Building, Harrisburg, Pa.

1905. RUGH, JAMES TORRANCE, M.D., Orthopedic Surgeon to the Methodist Hospital; Demonstrator of Orthopedic Surgery in the Jefferson Medical College; Assistant Orthopedist to the Jefferson Hospital; Assistant Orthopedist to the Philadelphia Hospital. 1616 Spruce St.

1897. SAILER, JOSEPH, M.D., Associate in Medicine in the University of Pennsylvania; Associate of the Pepper Clinical Laboratory of the University of Pennsylvania; Pathologist to the Pennsylvania Training School for Feeble-minded Children. 248 S. Twenty-first St.

1900. SAJOUS, CHARLES E. DE M., M.D. 2043 Walnut St.

1905. SARTAIN, PAUL J., M.D. 212 W. Logan Square.

1908. SAUTTER, ALBERT C., M.D., Assistant in the Dispensary for Diseases of the Eye in the University Hospital; Assistant in the Eye Dispensary of the German Hospital. 1421 Locust St.

1906. SAYLOR, EDWIN S., M.D., Ophthalmologist to St. Timothy's Hospital, Roxborough. 2005 Chestnut St.

1899. SCHAMBERG, JAY F., M.D., Professor of Dermatology and Infectious Eruptive Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine; Assistant Physician to the Municipal Hospital for Infectious Diseases. 1922 Spruce St.

1907. SCHWARTZ, GEORGE J., M.D., Assistant Surgeon to the Philadelphia Hospital; Instructor in Surgery in Jefferson Medical College. 1633 S. Fifteenth St.

1887. DE SCHWEINITZ, GEORGE E., M.D., Professor of Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Philadelphia Hospital; Consulting Ophthalmic Surgeon to the Philadelphia Polyclinic; Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1705 Walnut St.

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ELECTED

1895. Scott, J. Alison, M.D., Adjunct Professor of Medicine in the University of Pennsylvania; Visiting Physician to the Pennsylvania Hospital; Physician to the Church Home for Children. 1834 Pine St.

1892. Seiss, Ralph W., M.D., Professor of Otology in the Philadelphia Polyclinic; Consulting Laryngologist to the Pennsylvania Institution for the Deaf and Dumb. 255 S. Seventeenth St.

1908. Shannon, Charles E. G., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at Jefferson Medical College Hospital. 2025 Chestnut St.

1897. Sharpless, William T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1906. Shields, William G., M.D., Demonstrator of Morbid Anatomy in the Medico-Chirurgical College; Assistant Pathologist to the Philadelphia Hospital; Assistant Physician to the Howard Hospital; Assistant Physician to the Jewish Hospital. 412 School House Lane, Germantown.

1876. Shippen, Edward, A.M. (Princeton), M.D., Rear Admiral on the Retired List, U. S. Navy. 2039 Pine St.

1891. Shober, John B., A.M., M.D., Associate Gynecologist to the Gynecean Hospital; Gynecologist to the Howard Hospital; Obstetrician to the Philadelphia Hospital. 1731 Pine St.

1890. Shoemaker, George Erety, A.M., M.D., Gynecologist to the Presbyterian Hospital and to the Pennsylvania Epileptic Hospital and Colony Farm; Consulting Surgeon to the Woman's Hospital of Philadelphia. 1831 Chestnut St.

1908. Shoemaker, Harlan, A.B. (Stanford), M.D., Pathologist to the Methodist Hospital. 1618 Spruce St.

†1893. Shoemaker, Harvey, M.D., Visiting Physician to the Sheltering Arms and to the Southern Home for Destitute Children; Assistant Physician to the German Hospital; Physician to the Out-patient Department of the German and the Pennsylvania Hospitals. 2011 Chestnut St.

†1896. Shoemaker, William T., M.D., Ophthalmic Surgeon to the Germantown Hospital; Assistant Ophthalmologist and a Chief of Clinic to the German Hospital; Dispensary Ophthalmic Surgeon to the Presbyterian Hospital; Oculist to Pennsylvania Institution for the Deaf and Dumb. 2031 Chestnut St.

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ELECTED

1900. SHUMWAY, EDWARD ADAMS, B.S., M.D., Instructor in Ophthalmology in the University of Pennsylvania, and Assistant Ophthalmic Surgeon to the University Hospital; Assistant Ophthalmologist to the Philadelphia Hospital; Assistant Ophthalmologist and a Chief of Clinic to the German Hospital; Ophthalmologist to St. Vincent's Home. 2007 Chestnut St.

1903. SINCLAIR, JOHN FALCONER, M.D., Physician to the Medical Dispensary of the Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. SINKLER, FRANCIS WHARTON, A.B., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Physician to the Episcopal Hospital. 220 S. Sixteenth St.

1872. SINKLER, WHARTON, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases; Neurologist to the State Asylum for the Chronic Insane of Pennsylvania. 1606 Walnut St.

1902. SITER, E. HOLLINGSWORTH, M.D., Instructor in Genito-urinary Diseases in the University of Pennsylvania; Genito-urinary Surgeon to the Philadelphia Hospital; Surgeon-in-Charge of the Genito-urinary Dispensary of the University Hospital; Surgeon to the Dispensary of the Children's Hospital. 2038 Locust St.

1904. SKILLERN, PENN GASKILL, M.D. 241 S. Thirteenth St.

1895. SLOCUM, HARRIS A., M.D., Professor of Gynecology in the Philadelphia Polyclinic; Gynecologist to St. Clement's Hospital for Epileptics. 1900 Chestnut St.

1904. SMITH, ALLEN J., M.D., Professor of Pathology in the University of Pennsylvania; Pathologist to the University Hospital, to the Philadelphia Hospital, and to the State Hospital for the Insane at Norristown, Pa. University of Pennsylvania.

1905. SMITH, S. MACCUEN, M.D., Professor of Otology in the Jefferson Medical College; Aurist and Laryngologist to the Germantown Hospital; Aurist to the Jewish Hospital; Consulting Aurist to the Oncologic Hospital. 1429 Spruce St.

1908. SPEESE, JOHN, M.D., Assistant Instructor in Surgery in the University of Pennsylvania; Surgeon to the Dispensary of the University and the Children's Hospitals. 328 S. Sixteenth St.

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ELECTED

1895. SPELLISSY, JOSEPH M., A.M., M.D., Surgeon to the Outpatient Department of the Pennsylvania Hospital; Visiting Surgeon to St. Joseph's and to the Methodist Episcopal Hospitals; Assistant Surgeon to the Orthopædic Department of the University Hospital. 110 S. Eighteenth St.

1897. SPILLER, WILLIAM G., M.D., Associate Professor of Neurology and Professor of Neuropathology in the University of Pennsylvania; Clinical Professor of Nervous Diseases in the Woman's Medical College of Pennsylvania; Professor of Nervous Diseases in the Philadelphia Polyclinic and College for Graduates in Medicine. 4409 Pine St.

1908. SPITZKA, EDWARD ANTHONY, M.D., Professor of General Anatomy in the Jefferson Medical College. Jefferson Medical College.

1894. STAHL, B. FRANKLIN, B.S., Ph.G., M.D., Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Lecturer on Dietetics of the Sick and Instructor in Physical Diagnosis in the University of Pennsylvania; Visiting Physician to St. Agnes's Hospital; Visiting Physician to the Philadelphia Hospital. 1727 Pine St.

1904. STANTON, WILLIAM B., M.D., Physician to the Department of Tuberculosis, Philadelphia Hospital; Physician to the Henry Phipps Institute; Visiting Physician to the Free Hospital for Poor Consumptives, White Haven, Pa. 732 Pine St.

1875. STARR, LOUIS, M.D., LL.D. (Haverford). 1818 S. Rittenhouse Square.

1892. STEINBACH, LEWIS W., M.D., Professor of Clinical and Operative Surgery in the Philadelphia Polyclinic; Visiting Surgeon to the Philadelphia and to the Jewish Hospitals. 1309 N. Broad St.

1884. STELWAGON, HENRY W., M.D., Ph.D., Professor of Dermatology in the Jefferson Medical College; Dermatologist to the Philadelphia and to the Howard Hospitals; Associate Member of the Society of Dermatology and Syphilology of France and of Italy. 1634 Spruce St.

1895. STENGEL, ALFRED, M.D., Professor of Clinical Medicine in the University of Pennsylvania; Physician to the University, to the Pennsylvania, and to the Philadelphia Hospitals. 1811 Spruce St.

1901. STEVENS, ARTHUR A., M.D., Professor of Pathology in the Woman's Medical College of Pennsylvania; Lecturer on

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ELECTED

Physical Diagnosis in the University of Pennsylvania; Physician to the Episcopal and to St. Agnes's Hospitals. 314 S. Sixteenth St.

1902. STEWART, FRANCIS T., M.D., Professor of Surgery in the Philadelphia Polyclinic and College for Graduates in Medicine; Surgeon to the Germantown Hospital; Assistant Surgeon to the Jefferson Medical College Hospital; Surgeon to the Out-patient Department of the Pennsylvania Hospital. 311 S. Twelfth St.

1898. STOUT, GEORGE C., M.D., Professor of Otology in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society, and to the William Penn Charter School. 1611 Walnut St.

1884. STRYKER, SAMUEL S., M.D., Physician to the Presbyterian Hospital. 3833 Walnut St.

1900. SWAN, JOHN M., M.D., Instructor in Clinical Pathology, and in Tropical Medicine in the Philadelphia Polyclinic and College for Graduates in Medicine. 3713 Walnut St.

1898. SWEET, WILLIAM M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Associate Professor of Ophthalmology in the Jefferson Medical College; Assistant Ophthalmic Surgeon to the Philadelphia Hospital; Consulting Ophthalmologist to the Phœnixville Hospital. 1205 Spruce St.

1906. TAIT, THOMAS W., M.D., Ophthalmologist to St. Agnes's Hospital and to the Charity Hospital. 320 S. Fifteenth St.

1900. TALLEY, JAMES ELY, A.B., M.D., Physician to the Out-patient Department of the Presbyterian Hospital; Assistant and Consulting Physician to the Country Branch of the Children's Hospital. 1927 Chestnut St.

1901. TAYLOR, J. GURNEY, M.D., Physician to the Pennsylvania Institution for the Instruction of the Blind. 6041 Drexel Road, Overbrook, Pa.

1886. TAYLOR, JOHN MADISON, A.M. (Princeton), M.D., Editor *Monthly Cyclopedia of Practical Medicine;* Pediatric Physician to the Philadelphia Hospital; Assistant Physician to the Children's Hospital; Consulting Physician to the Elwyn, Pa., and to the Vineland, N. J., Training Schools for Feeble-minded Children. 1504 Pine St.

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ELECTED

1887. TAYLOR, WILLIAM J., M.D., Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases, and to St. Agnes's Hospital; Consulting Surgeon to the West Philadelphia Hospital for Women. 1825 Pine St.

1886. TAYLOR, WILLIAM L., M.D. 1340 N. Twelfth St.

1867. THOMAS, CHARLES HERMON, M.D. 3634 Chestnut St.

1907. THOMAS, THOMAS TURNER, M.D., Instructor in Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Assistant Surgeon to the Philadelphia Hospital. 2005 Chestnut St.

1897. THOMSON, A. G., M.D., Assistant Ophthalmic Surgeon to the University Hospital; Assistant Ophthalmologist to the Orthopædic Hospital and Infirmary for Nervous Diseases. 1426 Walnut St.

1896. THORINGTON, JAMES, A.M., M.D., Professor of Diseases of the Eye in the Philadelphia Polyclinic; Member of the American Ophthalmological Society; Ophthalmologist to the Vineland and Elwyn Training Schools for Feeble-minded Children. 120 S. Eighteenth St.

1898. THORNTON, EDWARD Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 922 Spruce St.

1896. TOULMIN, HARRY, M.D., Assistant Medical Director of the Penn Mutual Life Insurance Company. 925 Chestnut St.

1908. TRACY, STEPHEN E., M.D., Gynecologist to the Stetson Hospital. 1429 Spruce St.

1901. TUCKER, HENRY, M.D. 2000 Pine St.

†1894. TUNIS, JOSEPH PRICE, M.D., Clinical Assistant to the Nose and Throat Dispensary of the Polyclinic Hospital. 2045 Chestnut St.

1901. TURNER, JOHN B., M.D., Clinical Assistant in the Ophthalmological Out-patient Department of the Jefferson Medical College Hospital. 1833 Chestnut St.

1866. TYSON, JAMES, M.D., LL.D., Professor of Medicine in the University of Pennsylvania and Physician to the Hospital of the University of Pennsylvania; Physician to the Pennsylvania Hospital. 1506 Spruce St.

1897. TYSON, T. MELLOR, M.D., Physician to the Philadelphia Hospital; Assistant Physician to the Hospital of the University of Pennsylvania; Physician to the Rush Hospital, to the Philadelphia Lying-in Charity Hospital, and to the Children's Aid Society of Philadelphia. 1506 Spruce St.

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ELECTED

1904. UHLE, ALEXANDER A., M.D.; Assistant Instructor in Genito-urinary Diseases in the University of Pennsylvania; Assistant Surgeon to the Dispensary for Genito-urinary Diseases, University Hospital; Assistant Genito-urinary Surgeon to the Philadelphia Hospital; Assistant Surgeon to the Dispensary of the German Hospital. 1327 Jefferson St.

1907. ULLOM, JOSEPHUS TUCKER, A.B., M.D., Member of the Staff of the Henry Phipps Institute for Tuberculosis. 24 Carpenter St., Germantown.

1873. VAN HARLINGEN, ARTHUR, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic; Dermatologist to the Children's Hospital. 1831 Chestnut St.

1903. VAN PELT, WILLIAM TURNER, M.D., Ophthalmic Surgeon to the Episcopal Hospital. 1528 Spruce St.

1893. VANSANT, EUGENE LARUE, M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic; Visiting Physician to the Throat, Nose, and Ear Department of the Howard Hospital. 1929 Chestnut St.

*1897. VEASEY, CLARENCE A., A.M., M.D., Suite 205, Traders' Bank Building, Spokane, Wash.

†1883. VINTON, CHARLES HARROD, M.D. Wernersville, Berks County, Pa.

1903. WADSWORTH, WILLIAM SCOTT, M.D. The Normandie, Thirty-sixth and Chestnut Sts.

1885. WALKER, JAMES B., M.D., Ph.D., Consulting Physician to the West Philadelphia Hospital for Women and Children. 1617 Green St.

1906. WALKER, JOHN K., M.D., Physician to the Dispensary of the Mary J. Drexel Home. 1632 Spruce St.

1907. WALKER, WARREN, M.D., Surgeon to the Dispensary of the Episcopal Hospital; Surgeon to the Dispensary of the Children's Hospital; Assistant Surgeon in the Clinic for Genito-urinary Diseases of the Polyclinic Hospital. 1632 Spruce St.

1904. WALSH, JOSEPH, A.M., M.D., Physician to the Henry Phipps Institute; Visiting Physician to the White Haven Sanatorium; Physician to the Department of Tuberculosis at the Philadelphia Hospital. 732 Pine St.

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ELECTED

1893. WARREN, JOSEPH W., M.D., Associate Professor of Physiology in Bryn Mawr College. Bryn Mawr, Pa.

1895. WATSON, ARTHUR W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to the Home for Incurables. 126 S. Eighteenth St.

1886. WATSON, EDWARD W., M.D., Physician to the Magdalene Home. 38 S. Nineteenth St.

1903. WEBER, CHARLES H., M.D., Physician to the Episcopal Hospital; Physician to the Dispensary of the Children's Hospital. 1304 Pine St.

1906. WEISENBURG, THEODORE H., M.D., Professor of Neuropathology and Associate Professor of Neurology in the Medico-Chirurgical College; Associate in Neurology in the Philadelphia Polyclinic; Neurologist to the Philadelphia Hospital. 2030 Chestnut St.

1883. WELCH, WILLIAM M., M.D., Physician-in-Charge of the Municipal Hospital for Contagious Diseases; Consulting Physician to the Northern Dispensary and to the Northern Home for Friendless Children. 1411 Jefferson St.

1897. WELLS, WILLIAM H., M.D., Associate in Obstetrics in the Jefferson Medical College; Assistant Obstetrician to the Jefferson Medical College Hospital. 1105 Spruce St.

1893. WESTCOTT, THOMPSON S., M.D., Associate in Diseases of Children in the University of Pennsylvania; Visiting Physician to the Methodist Episcopal Hospital; Assistant Physician to the Children's Hospital; Pediatrist to the Jewish Hospital. 1720 Pine St.

1884. WHARTON, HENRY R., M.D., Clinical Professor of Surgery in the Woman's Medical College of Pennsylvania; Surgeon to the Presbyterian and to the Children's Hospitals; Consulting Surgeon to the Bryn Mawr Hospital, to St. Christopher's Hospital for Children, and to the Pennsylvania Institution for the Deaf and Dumb. 1725 Spruce St.

1901. WHITE, COURTLAND Y., M.D., Lecturer on and Demonstrator of Gross Morbid Anatomy in the Veterinary Department of the University of Pennsylvania; Pathologist to the State Live Stock Sanitary Board, to the Children's and to St. Joseph's Hospitals; Pathological Director at the Episcopal Hospital. 1808 Diamond St.

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ELECTED

1878. WHITE, J. WILLIAM, M.D., Ph.D., LL.D. (Hon. Aberdeen), John Rhea Barton Professor of Surgery in the University of Pennsylvania; Senior Surgeon to the University Hospital; Fellow of the American Surgical Association and of the American Association of Genito-Urinary Surgeons. 1810 S. Rittenhouse Square.

1905. WHITEWAY, HAROLD M., M.D. 1924 Chestnut St.

1898. WHITING, ALBERT D., M.D., Surgeon to the Germantown Hospital; Assistant Surgeon to the German Hospital; Surgeon to the Southern Home for Destitute Children; Surgeon to the Out-patient Department of the German Hospital. 1523 Spruce St.

†1880. WILLARD, DE FOREST, A.M., M.D., Ph.D., Professor of Orthopedic Surgery in the University of Pennsylvania; Surgeon-in-Chief to the Widener School; Consulting Surgeon to the Presbyterian, to the Germantown, to the Jewish, to the Atlantic City, and to the Phœnixville Hospitals. 1901 Chestnut St.

1907. WILLIAMS, CARL, B.S., M.D., Assistant Ophthalmic Surgeon to the Germantown Hospital; Assistant Surgeon to the Dispensary for Diseases of the Eye at the University Hospital. 5909 Germantown Ave.

1878. WILLIAMSON, JESSE, M.D. 8708 Germantown Ave., Chestnut Hill.

1902. WILLSON, ROBERT N., JR., M.D., Instructor in Physical Diagnosis, and Students' Physician in the University of Pennsylvania. 1708 Locust St.

1881. WILSON, H. AUGUSTUS, M.D., Professor of Orthopedic Surgery in the Jefferson Medical College; Emeritus Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Consulting Orthopedic Surgeon to the Philadelphia Lying-in Charity Hospital, and to the Kensington Hospital for Women; Senior Orthopedic Surgeon to the Philadelphia Hospital. 1611 Spruce St.

1874. WILSON, JAMES C., A.M. (Princeton), M.D., Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College, and Physician to the Hospital of the same (Faculty Staff); Physician-in-Chief to the German Hospital; Attending Physician to the Pennsylvania Hospital. 1509 Walnut St.

1902. WILSON, SAMUEL M., M.D. 1517 Arch St.

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ELECTED

1897. WILSON, W. REYNOLDS, M.D., Visiting Physician to the Philadelphia Lying-in Charity Hospital. 1709 Spruce St.

1904. WISTER, JAMES W., M.D., Physician to the Out-patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. WITMER, A. FERREE, M.D. Freeport, Long Island, N. Y.

1893. WOOD, ALFRED C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Assistant Surgeon to the University Hospital; Surgeon to the Philadelphia and to St. Timothy's Hospitals. 128 S. Seventeenth St.

1900. WOOD, GEORGE B., M.D., Instructor in Laryngology in the University of Pennsylvania; Assistant in the Clinic for Diseases of the Nose and Throat at the Polyclinic Hospital. 129 S. Eighteenth St.

1865. WOOD, HORATIO C., M.D., LL.D. (Yale and Lafayette), Emeritus Professor of Materia Medica and Therapeutics in the University of Pennsylvania; Associate Fellow in Medicine and Surgery of the American Academy of Arts and Sciences; Member of the National Academy of Science. 4107 Chester Ave.

1903. WOOD, HORATIO C., JR., M.D., Associate Professor of Pharmacology in the University of Pennsylvania. 434 S. Forty-fourth St.

1907. WOOD, WALTER A., M.D. 1715 Spruce St.

1880. WOODBURY, FRANK, M.D., Secretary to the Committee on Lunacy of the Board of Charities of Pennsylvania. 218 S. Sixteenth St.

1866. WOODS, D. F., M.D., Physician to the Presbyterian Hospital. 1501 Spruce St.

1901. WOODS, RICHARD F., M.D., Assistant Surgeon to the Gynecean Hospital. 1501 Spruce St.

1888. WOODWARD, CHARLES E., M.D., Secretary of the West Chester Board of Health; U. S. Examining Surgeon; Member of the Medical Staff of the Chester County Hospital. West Chester, Pa.

†1897. WOODWARD, GEORGE, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. WORDEN, CHARLES B., M.D., Associate in Diseases of the Stomach and Intestines in the Philadelphia Polyclinic; Physician to the Dispensary of the Presbyterian Hospital; Anesthetizer to the Orthopedic Department of the University Hospital; Physician to the Presbyterian Orphanage. 322 S. Sixteenth St.

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ELECTED

1889. YOUNG, JAMES K., M.D., Professor of Orthopedic Surgery in the Philadelphia Polyclinic; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Associate in Orthopedic Surgery in the University of Pennsylvania and Assistant Orthopedic Surgeon to the University Hospital. 222 S. Sixteenth St.

1894. ZENTMAYER, WILLIAM, M.D., Attending Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to St. Mary's Hospital; Ophthalmologist to the House of Refuge. 1819 Spruce St.

1899. ZIEGLER, S. LEWIS, A.M., M.D., Sc.D., Attending Surgeon to the Wills Eye Hospital; Ophthalmic Surgeon to St. Joseph's Hospital; Membre Société Française d'Ophtalmologie. 1625 Walnut St.

1887. ZIEGLER, WALTER M. L., A.M., M.D. 1418 N. Seventeenth St.

1895. ZIMMERMAN, MASON W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1522 Locust St.

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ASSOCIATE FELLOWS.

[Limited to Fifty, of whom Twenty may be Foreigners.]

AMERICAN.

ELECTED

1876. BILLINGS, JOHN S., M.D., U. S. A., Astor Library Building, 40 Lafayette Place, New York City, New York.

1886. BOWDITCH, HENRY P., M.D., 688 Boylston Street, Boston, Massachusetts.

1877. CHAILLE, STANFORD E., M.D., University Building, New Orleans, Louisiana.

1886. CHEEVER, DAVID W., M.D., 557 Boylston Street, Boston, Massachusetts.

1896. CONNER, PHINEAS SANBORN, M.D., 215 West Ninth Street, Cincinnati, Ohio.

1893. COUNCILMAN, WILLIAM T., M.D., Harvard Medical College, Boston, Massachusetts.

1892. EMMET, THOMAS ADDIS, M.D., 91 Madison Avenue, New York City, New York.

1892. FITZ, REGINALD H., M.D., 81 Arlington Street, Boston, Massachusetts.

1895. FLETCHER, ROBERT, M.D., Army Medical Museum, Washington, D. C.

1903. GORGAS, WILLIAM C., M.D., U. S. A., Washington, D. C.

1891. JACOBI, A., M.D., 19 East Forty-seventh Street, New York City, New York.

1906. MAYO, WILLIAM J., M.D., Rochester, Minn.

1895. MCBURNEY, CHARLES, M.D., 28 West Thirty-seventh Street, New York City, New York.

1906. PILCHER, LEWIS STEPHEN, M.D., 386 Grand Avenue, Brooklyn, New York.

1886. REEVE, JOHN C., M.D., LL.D., S. W. corner Third and Wilkinson Streets, Dayton, Ohio.

1906. SHATTUCK, FREDERICK C., M.D., 135 Marlborough Street, Boston, Massachusetts.

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ELECTED

1896. STERNBERG, GEORGE M., M.D., U. S. A., 1019 Sixteenth Street, N. W., Washington, D. C.
1896. TIFFANY, L. MCLANE, M.D., 831 Park Avenue, Baltimore, Maryland.
1894. WARREN, J. COLLINS, M.D., 58 Beacon St., Boston, Massachusetts.
1894. WEIR, ROBERT F., M.D., 11 East Fifty-fourth Street, New York City, New York.
1892. WELCH, WILLIAM H., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

FOREIGN.

1890. BACCELLI, GUIDO, Rome, Italy.
1908. BANNERMAN, W. B., M.D., Lt. Col. I. M. S., 11 Strathearn Place, Edinburgh, Scotland.
1894. BRUNTON, SIR T. LAUDER, M.D., 10 Stratford Place, London, W., England.
1903. FINLAY, CHARLES J., M.D., Havana, Cuba.
1899. FRASER, SIR THOMAS R., M.D., LL.D., F.R.C.P., F.R.S., 13 Drumsheigh-Gardens, Edinburgh, Scotland.
1903. HORSLEY, SIR VICTOR ALEXANDER HADEN, F.R.S., 25 Cavendish Square, London, England.
1906. HUTCHINSON, JONATHAN, M.D., LL.D., F.R.S., 15 Cavendish Square, London, W., England.
1896. JACCOUD, PROF. S., Rue Tronchet 35, Paris, France.
1894. JACKSON, J. HUGHLINGS, M.D., 3 Manchester Square, London, England.
1893. VON JAKSCH, RUDOLF, M.D., Prague, Bohemia.
1903. KOCHER, PROF. THEODOR, M.D., Berne, Switzerland.
1896. LEYDEN, ERNST, M.D., Berlin, Germany.
1877. LORD LISTER, M.D., LL.D., F.R.S., 12 Park Crescent, Portland Place, London, W., England.
1906. MYLES, SIR THOMAS, M.D., 33 Merion Square, W., Dublin, Ireland.
1896. PYE-SMITH, P. H., M.D., 48 Brook St., London, W., England.
1898. RODDICK, THOMAS G., M.D., 80 Union Avenue, Montreal, Canada.
1908. ROSS, MAJOR RONALD, M.D., R.A., Liverpool School of Tropical Medicine, Liverpool, England.
1869. VALCOURT, TH. DE, M.D., Cannes, France.
1904. WALDEYER, PROF. WILHELM, M.D., Berlin, Germany.

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CORRESPONDING MEMBERS.

ELECTED

1880. CARROW, FLEMMING, M.D., Washington Arcade, Detroit, Michigan.

1880. CHIARA, DOMENICO, M.D., Florence, Italy.

1886. DEY, KANNY LALL, M.D., Calcutta, India.

1885. RENDU, JEAN, M.D., Lyons, France.

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NECROLOGICAL LIST.

FELLOWS.

HENRY D. BENNER,	December 8, 1907
LEONARDO DA VINCI JUDD,	March 18, 1908
J. DUTTON STEELE,	May 17, 1908
HORACE Y. EVANS,	July 23, 1908

ASSOCIATE FELLOWS.

NICHOLAS SENN,	January 2, 1908
FELIX TERRIER,	April 18, 1908

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ANNUAL ADDRESS OF THE PRESIDENT.[1]

By JAMES TYSON, M.D.

Few will deny that the most important event in the history of the College in the past year was the decision to dispose of our present site and build a new hall on the recently acquired lot on the west side of Twenty-second Street, below Chestnut, at the corner of Ludlow.

After much seeming vacillation, which was the result of an undoubted desire on the part of the Fellows to further the best interests of the College, at the stated meeting in March, when 178 Fellows registered their names, the decision in favor of removal, previously reached and again set aside, was re-affirmed by a very large majority. This action was followed by an order from the College to an existing Building Committee to select architects and secure plans, specifications, and estimates, a preliminary appropriation of $3000 being made for expenses. Later, the College authorized a total expenditure not exceeding $250,000.

After many months of careful and conscientious work plans, which were freely submitted to the Fellows for criticism, have been agreed upon, and the committee is encouraged to believe the building can be constructed for little more than the sum to which expenditure was limited by the College, $250,000.

As to other transactions of the College, business and scientific, they have been in no way startling, partly because of the deep absorption of our interest in the matter of the new building, on account of which other business was at times formally discouraged. The sections, however, especially that of Medicine, have made

[1] Read January 1, 1908.

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up for the deficiency. It is much to be regretted that the papers read before the sections do not appear in the TRANSACTIONS, because our income is inadequate to meet the necessary cost. A sufficient publication fund is much needed by the College. The provision of a Committee on Scientific Business to look after suitable papers for the general meetings is expected to improve the quality and suitableness of subjects to be treated at those meetings.

A melancholy circumstance in the history of the past year is the unusual large necrology, both as to the eminence and number of the dead. Never in a single year in the recollection of the President have so many well-known Fellows joined the procession to the unknown country.

They included:

1. C. Percy La Roche, who died in Rome, March 12, 1907. He was elected a Fellow in July, 1865. He was a polished and intelligent gentleman, who reflected in his manners and deportment the effect of long residence abroad and association with the best of his class. He practised medicine little or none in this country.

2. John H. Brinton died March 18, 1907. He was elected a Fellow in October, 1856. He early became distinguished in the Civil War by his executive and literary ability, and he was one of the founders of the United States Army Medical Museum, which today ranks among the finest museums in the world. I was fortunate to be assigned for a short time at Winchester, Va., in the fall of 1864, when he too was stationed there, and I recall with pleasure his appearance as he galloped through the town on some tour of duty. He was Chairman of the Mütter Museum Committee for many years, and conspicuous among the regular attendants at our meetings.

3. John H. Packard, who died on May 21, 1907, was elected a Fellow in July, 1858. He was no less conspicuous in the history of the College. For many years he was Secretary and managed its affairs skilfully. He arose to the position of Vice-President, and should have been President. His skill in drawing enabled him to illustrate with facility, and his note books were copiously

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illustrated. His knowledge of art caused him to be continued for years on the Board of Directors of the Academy of Fine Arts of Philadelphia.

4. Charles E. Cadwallader, elected a Fellow in March, 1886, died in London, in June, 1907. He was a member of a distinguished family. At one time on the staff of General Meade, he, too, represented the profession in the War of the Rebellion. He was a well-educated physician, who preferred to practise among the poor, and declined fees from them.

5. James W. MacIntosh, elected April, 1905, died in June, 1907.

6. Frank H. Getchell died June 27, 1907. He was elected a Fellow in July, 1864. Born in Maine, he entered the Union army at the breaking out of the Rebellion, as surgeon to a Maine regiment. After serving a term of service he located in Philadelphia, and was attached to U. S. A. Hospitals until the close of the war. He was for a time lecturer on gynecology in the Jefferson Medical College.

7. William Thomson died in August, 1907, having been chosen a Fellow in April, 1869. He was one of the most eminent of the Fellows of the College. He entered the regular army at the opening of the Civil War, and his ability as an organizer was promptly recognized. He was surgeon in charge of one or more military hospitals, and assisted in the organization of others. He was one of the first three Philadelphians who secured for ophthalmology its recognition as an acknowledged specialty, demanding the most thorough scientific training of those who attempt its practice. The two ophthalmologists whose names were linked with his were Dr. Ezra Dyer and Dr. Wm. F. Norris.

8. Charles Stewart Wurtz died on August 13, 1907, having been elected a Fellow in October, 1860. This refined and courteous gentleman never practised medicine actively, but was very glad to keep up his connections with the profession by serving the College for many years as its Treasurer. He filled other positions of honor and trust in the community.

9. William G. B. Harland was one of the younger Fellows of the College, having been elected in February, 1902, and died

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Septemper 14, 1907, of appendicitis. He was acquiring eminence as a laryngologist and throat specialist, when his untimely death cut short his career.

10. William Moss was elected a Fellow in October, 1864, and died in October, 1907. Dr. Moss was an accomplished physician and refined, scholarly gentleman. He, too, served in the Civil War with distinction, and commanded the respect of all who had to do with him. He had, until his health began to decline, a large practice at Chestnut Hill among the most influential residents of that suburb.

11. Dr. Henry Benners, elected a Fellow in 1860, died December 8, 1907. He was a highly respected practitioner who had for many years a large general practice in the southern part of the city.

12. Robert Barnes, of London, was an Associate Fellow, elected in 1877, and died in May, 1907. His writings on obstetrics and gynecology place his name among the first of English eminent physicians.

13. Sir Joseph Fayrer, of London, was elected in 1883, and died in May, 1907. He is best known by researches on snake venom made between 1868 and 1884. He also wrote numerous papers on pathology and surgery, and received many recognitions of his merits by degrees conferred and by election to learned societies.

Thus eleven Fellows and two Associate Fellows were retired by death from our rolls, while five Fellows resigned, being non-resident men, some of whom were probably influenced by the new law which requires the non-resident Fellows to pay $5.00 annually.

Twenty-eight Fellows were added and one Associate Fellow, making the roll as follows: 429 Fellows, of whom 398 are resident in the city or near vicinity, and 31 non-resident; 40 Associate Fellows, of whom 22 are Americans and 18 foreign; 4 Corresponding Members, making a grand total of 473 and a net gain of only 11 for the year.

Whence, too, it is evident that our membership is increasing slowly, and too slowly if our College is to continue to exert an influence proportionate to its age and traditions. That our roll may be enlarged by the addition of desirable members I am confident.

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There are many reputable physicians within our city limits who would not think of suggesting their nomination who should be sought and invited to join. In fact, the most desirable Fellows are those whose qualifications and good breeding make them hesitate to express a desire for membership. I do not like the expression "applications for fellowship." There should be no such phraseology, and there is none in our printed forms. Our forms are "nominations to Fellowship" made by a legal number of Fellows, who should see to it that the nominee is appropriately represented to the Council to secure his election. I believe that at least twenty-five reputable physicians thoroughly eligible and willing to become Fellows could be found in a short time if the proper measures are taken. On the other hand, men who seek membership are not always those who most grace our roll, or are even desirable.

The mode in which the accounts of the College are kept is unique and correspondingly difficult to grasp or comprehend by the average mind. Efforts have been made to correct the mystifying methods through Committees, but have thus far failed. A part of the difficulty appears to be in the conditions under which some donations have been made to the College, but there ought to be a way out of it. If necessary, an expert accountant might be engaged to help us. I am in favor of publishing also annually the report of the Treasurer. Nothing is so difficult to follow mentally as figures, and it would be very helpful to Fellows if they could have laid before them a printed copy of the Treasurer's report, by which they could follow him intelligently.

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ERYSIPELOID OF ROSENBACH:[1]

A NOTE ON ITS OCCURRENCE IN LABORATORY WORKERS.

By JOHN H. JOPSON, M.D.

My object in making this report is to place on record two cases of this interesting and somewhat unusual affection, contracted in an unusual manner, as well as to make mention of a third case, which, while not seen personally, had symptoms, as related to me, that were strongly suggestive of this affection. These three cases were all in scientific investigators or their assistants, engaged in studies in comparative and experimental pathology.

E. H. G., a physician in excellent health, pricked the ring finger of the left hand, or infected a previously existing abrasion in that locality, while working with a mass of semiputrid pigs' snouts, from which he was making chondro-sulphuric acid to be used in the experimental production of amyloid disease. Within thirty-six hours he developed what appeared to be a cellulitis of the end of the finger, and on the following day, December 9, consulted me.

The appearance was that of an ordinary paronychial affection. It was incised, but no pus was found. The swelling and tension were relieved, and in a few days it was apparently well.

He returned to his previous work, protecting the finger in a measure by a finger-cot. Within twenty-four hours, December 14, he noticed a swelling near the end of the finger. This swelling was tense, dark red, and limited by a well-defined margin, without vesiculation or pustulation, and differing entirely from an ordinary cellulitis in appearance. I was able to make a diagnosis of erysipeloid at once. An ichthyol ointment dressing was applied.

The infection spread slowly toward the base of the finger, accompanied by itching and burning, but without constitutional disturbance. Iodine was now applied around the infected area and beyond, but without effect in checking

[1] Read January 1, 1908.

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its spread. On the 19th it was noticed that it had spread to the palm of the hand. Lead-water and laudanum dressings were used. Three or four days later the swollen and red area began to fade.

The whole course of the infection was about fourteen days from the time of the first symptoms of cellulitis. It is possible that there were two infections of different nature, the erysipeloid being secondary to the primary cellulitis.

Miss P., an assistant in the Laboratory of Comparative Pathology at the Zoölogical Gardens, and a young adult in good health, on October 28, 1907, while performing an autopsy on a cassowary that had died of acute enteritis, cut herself in the palmar surface of the left index finger while opening the intestine with a pair of scissors, but without drawing blood. Shortly afterward she washed the wound with carbolic acid and went on working. Later, she washed it with soap and water, lysol, and alcohol.

On the following day, without protecting this abrasion, she posted a Dorcas goat that had died of tuberculosis. On the evening of the same day she noted a swelling with inflammation at the site of the wound. There was no special pain. The inflammation travelled toward the base of the finger. The finger became red and swollen, and the seat of burning pain. She applied a poultice, and the inflammation receded toward the tip of the finger. On the 31st it returned to the middle phalanx.

She continued her poultices for three days, when the dressing was changed to hot carbolic, 1 to 100. Later, hot bichloride solution was used.

When I saw her, on November 5, 1907, the finger was swollen and slightly reddened. There were signs of an old abrasion over the second joint. The swelling was most marked at the proximal phalanx. The skin had been somewhat bleached by the hot, moist dressings. There was slight tenderness, but only a little pain; slight edema, but no fluctuation. The temperature was 99.1°. There had been headache and slight feverishness the previous evening. A diagnosis of erysipeloid was made, and lead-water and laudanum applied.

Two days later the swelling and redness were around the base of the finger, and had spread in the characteristic manner to a slight extent to the palm, with the usual well-defined margin marking its progress. There was a small blister on the dorsum of the finger, near the base, probably due to the application used. An ichthyol dressing was applied.

Cultures were made from the fluid in the blister by Dr. Herbert Fox, on glycerin-blood agar, saccharose agar, and glucose agar.

November 9, two days after this, the inflamed area had faded. There was no more pain. Burning and itching had been the most prominent symptoms. Three days later, November 12, it was noted that the inflammation had gone back to the end of the finger, which was red and swollen, with some throbbing, itching, and burning. After this the condition quickly subsided, and three days later she was practically well. The cultures failed to show a growth.

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Dr. H. F., a young adult in good health, detailed to me, in a personal communication, the following history:

On the day before infection developed he had made a postmortem on a bird whose species he could not recall. He did not remember having had any break in the continuity of the skin. Infection began in the first phalanx of the thumb, which in eighteen hours became swollen, red, and tender, as well as somewhat stiff. Twenty-four hours later the inflammation had spread to the thenar, eminence, which was swollen and painful, and also itched and burned. The first region affected was less painful.

The following day the inflammation returned to the starting point, travelling thence to the distal phalanx. On about the tenth day the proximal phalanx again became swollen and inflamed. It was incised, but no pus was found on deep incision. There was, however, said to have been a small necrotic area. The wound was not packed. A wet dressing was applied, and it healed in about a week. The temperature was not taken at any time, but he thought that he had slight fever.

Erysipeloid of Rosenbach (also known as erythema migrans, erythema serpens, reticular lymphangitis, and crab cellulitis) was first clearly described by Morrant Baker in 1873, under the name of erythema serpens. Tilbury Fox had, three years before, placed on record two cases of what were perhaps examples of this disease, although the infection was derived, not from animal matter but from kid shoes in one case and from clothing in another.

Erythema serpens, as described by Baker, was an infection of the hands that he observed in the butchers of the Smithfield meat markets. His description of the manner of infection, the general appearance, the tendency to spread, the self-limited course, and the absence of severe constitutional symptoms, corresponds closely to what we now recognize as erysipeloid. There were some minor differences, such as the exact color of the lesion, the shape of its margin, etc.

Rosenbach first described it in his book on *Mikroorganismen bei dem Wund Infektionskrankheiten;* and later, in 1887, he published his paper on the subject in the *Arch. f. klin. Chir.* He gave to it the name that it now commonly bears, erysipeloid; pointed out the manner of infection, the common infecting agents, and the classes of persons exposed; described its clinical symptoms and course; and made certain bacteriological studies.

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Following him, Elliott, Cordua, Felsenthal, and Delbanco reported cases or made studies in the etiology. Finally, Gilchrist published a very large series of cases, with clinical, pathological, and bacteriological observations, from the surgical and dermatological clinics of the Johns Hopkins Hospital.

Briefly, erysipeloid is an erythematous inflammation, somewhat resembling erysipelas, observed usually on the hands of persons engaged in handling animal products that are liable to putrefaction and decay, especially fish, shell-fish, meats, poultry, and cheese. Hence it is commonly encountered in fish, game, and poultry dealers, butchers, and cooks.

A large number of the reported cases (323 of the 329 reported by Gilchrist) are due to crab bites or to lesions produced by handling crabs. This type differs slightly, if at all, from that resulting from infection from dead animal matter. The explanation that has occurred to me is that the crab, as is well known, is a feeder on decomposing animal matter. As crabs are handled in the market, they are liable to be contaminated by such matter; and, as the crab does its biting with its hands, as it were, with which it also handles its food, organisms may readily be conveyed thereby. It is not necessary to suppose, as does Gilchrist, the presence of any special ferment in the crab.

The only recorded cases in which infection has been contracted in a manner similar to that in the cases here reported by me are those mentioned by Shepherd in the discussion on Gilchrist's paper before the American Dermatological Association in June, 1904. He stated that he had seen several cases in medical students, contracted from scratches in the postmortem or dissecting room. This is the only reference that I have found to inoculation in members of our profession.

Cases of this disease are of comparative infrequency; or, at least, in the ordinary experience of dermatologists. Only four cases were observed in one year in Unna's entire clinic. The remarkable experience of Gilchrist and his associates at Johns Hopkins Hospital, whereby he was able to report 329 cases, is explained by the fact that 323 of these were lesions contracted in the handling of crabs.

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The number of men engaged in the handling and shipping of crabs in Baltimore must be taken into consideration. The dermatologists with whom I have conversed have seen a very limited number of cases.

That they are not more frequently reported admits of two explanations: one, that they are comparatively rare; the other, that they are frequently overlooked. My attention was first directed to this disease some years ago, while working in the surgical dispensary at the Episcopal Hospital, where I saw a small series of cases, mostly in handlers of fish and shell-fish. It is probable that in surgical dispensaries, at least, they are usually looked upon as variations of ordinary cellulitis. At the same time it must be admitted that they are comparatively rare.

The infection takes place through a break in the continuity of the skin. Several points of inoculation are occasionally observed. The incubation period, according to Gilchrist's observations, averages two days. It may develop after a few hours, however, and rarely is delayed for several days or a week.

It appears as a swelling with elevated, sharply defined edges, which soon affects the entire circumference of the finger, and is commonly described as of a dark-red color, with purplish or even livid edges. The finger is tense and only moderately painful, but itching and burning are prominent symptoms. Sections of tissue excised show an inflammation of the entire corium and, to a slight extent, of the subcutaneous tissue, with infiltration of polynuclear leukocytes and small lymph cells; and edema of the epithelial cells of the epiderm; and inflammatory changes, especially marked around the sweat glands and bloodvessels (Gilchrist).

It has a characteristic tendency to spread from its usual point of origin, near the end of the finger, toward the palm, the primarily affected area fading from red to yellow, and thence to normal. Reaching the palm, it may spread over it, but commonly it soon affects the neighboring finger, and, when untreated, it may gradually spread to all the fingers and to the back of the hand.

There is no fever or other constitutional disturbance, and the lymph glands are almost never involved. It is more or less self-

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limited, commonly lasting from ten days to three weeks, and during this time there is a well-marked tendency to relapse. There is no suppuration, pustulation, or vesiculation, and no scaling follows.

Rosenbach obtained a culture that grew on gelatin at a low temperature. In its early stages it resembled a staphylococcus, being somewhat larger and in the shape of irregularly rounded and long bodies, which were the first stages of a thread-producing microörganism. This later developed as numerous tangled threads, of irregular length, very slender, arranged now irregularly, and again in spirals, and forming a thick, tangled mass. It resembled a cladothrix. Inoculations of this culture on the human subject produced, according to Rosenbach, a typical lesion at the end of forty-eight hours.

Following Rosenbach, Cordua and Felsenthal confirmed Rosenbach's theories as to causation by a specific germ, a cladothrix; while Delbanco, working in Unna's clinic, failed absolutely to obtain the organism. Gilchrist reports the results of investigations by Finney, Wright, Abbot, Livingood, and himself. Their results in a series of most carefully conducted histological and bacteriological studies were absolutely negative. While Rosenbach's views regarding the causation of erysipeloid are still considered sound in some text-books, it is evident that his experiments were conducted so long ago, and that his results have so frequently failed of confirmation, that one may be pardoned for looking askance at them.

That erysipeloid is due to a specific infection is strongly indicated. The manner of infection, the pathological findings, and the clinical course are scarcely explainable on other grounds. But it certainly cannot yet be said that the specific germ has been demonstrated beyond a doubt.

Erysipeloid is commonly said to require differential diagnosis from erysipelas, ring-worm, and eczema; but a consideration of the local nature of the affection, the manner of infection, the clinical appearance, and the self-limited course will usually suffice to separate it from cases of the other affections mentioned. In a few cases it may be difficult to distinguish erysipeloid at its onset from

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an ordinary cellulitis due to pyogenic infection. In such a contingency, observation for two days will usually enable one to draw a distinction.

A number of methods of treatment have been tried. Gilchrist claims the best results from the use of Unna's salicylic acid plaster in 25 per cent. strength, applied over and beyond the infected area, and covered with ordinary adhesive strips. In other cases, pressure with adhesive plaster alone sufficed to cure. The injection of carbolic acid, 1 to 20 to 1 to 30, around the edge; soaking in bichloride solution, applications of tincture of iodine, ichthyol ointment, and lead-water and laudanum, have all been used. We have thought that tincture of iodine, painted around and beyond the advancing margin, had sometimes an influence in checking the spread of the condition. Lead-water and laudanum and ichthyol we have found comfortable and soothing dressings. Gilchrist's results with salicylic plaster excel any that we have obtained by other means.

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A CASE OF MULTIPLE, SACCULATED THORACIC ANEURYSM SUCCESSFULLY TREATED BY WIRING.[1]

By JAMES M. ANDERS, M.D., LL.D.,
AND
JAMES P. MANN, M.S., M.D. (by invitation).

The patient, M. W., a colored man, aged thirty-six years, was admitted to the Medico-Chirurgical Hospital by Dr. Mann and placed under my care on January 18, 1907; his principal complaint was of a painful, throbbing lump in the sternal region, a troublesome cough, marked dyspnea, and general weakness.

The family history was negative. As a child he had measles and mumps; when about ten years of age he fell and sustained an injury to the chest, to which accident he attributed his present condition, although it was twenty years later when the first local symptoms appeared. He had always been more or less subject to "colds," and had suffered from several slight attacks of acute rheumatism.

Prior to twenty-four years of age he had been employed for a number of years on a farm, while for the past twelve years his occupation has been that of a barber; he has been married eight years, has used tea and coffee in moderation, but tobacco, beer, and whisky freely until entering the hospital. The patient admits specific infection, the primary lesion appearing, however, only four months previous to the time of his admission.

The present illness began about four years ago, when he noticed a dull, boring pain under the sternum. Soon a small, painful, throbbing protuberance appeared to the right of the midsternal region, but this swelling largely disappeared under absolute rest and certain medical measures. During the four years intervening between that date and his admission to the hospital he had an occasional pain in the same region, and a cough at intervals, which, however, was not characteristic of thoracic aneurysm.

Since Christmas (1906) he has had a severe cough, which caused considerable pain in the area of the original swelling. On January 14 (four days prior

[1] Read February 5, 1908.

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to date of admission) he indulged himself in a "spree," during which he suddenly felt a sharp pain, accompanied by a sense of "something giving way," in the chest. This was attended with a smothering sensation, and at the same time he noticed a return of the swelling. The tumor now rapidly enlarged, and four days later it was about the size of a small orange. There was neither dysphagia nor aphonia present.

Upon admission to the hospital, the most striking feature of the physical examination was a conical tumor mass projecting from the thorax at the right edge of the middle of the sternum. This was about two and one-half inches in height and three inches in diameter at the base. The apex showed a subcutaneous hemorrhage, which rapidly increased in extent until it was the size of a silver half-dollar. The extreme summit of this area was of a deep-red color and presented a glazed appearance. A pulsation was both visible and palpable; this was expansile in character, and extended for a considerable distance beyond the base of the mass. There was an absence of thrill, tracheal tugging, and episternal pulsation. A systolic murmur was heard at the second aortic interspace during inspiration only; this was transmitted into the vessels of the neck, but was not audible over the elevation. The heart was apparently neither enlarged nor displaced. The heart sounds at the apex were somewhat rapid and rather weak, the second aortic sound, however, was moderately accentuated. The lungs were normal excepting slight congestion of the lower portion of the left lung.

The pupils were equal and reacted to light, accommodation, and convergence. Estimation of the blood pressure in both radials showed the right to be systolic 130 mm. Hg., left 125 mm. Hg.; later, this difference was more marked, viz., right 120 mm., left 100 mm.

An *x*-ray examination, made by Dr. Pfahler immediately after the patient entered the hospital, showed an aneurysm extending three and one-half inches to the right of the median line on a level with the third rib and also approximately two and one-half inches upward and downward from this level. The upper portion of the aneurysmal shadow just overlapped the origin of the aorta.

The blood showed a slight grade of secondary anemia; coagulation time, three and one-half minutes; later (June 7) this was reduced to two minutes.

A diagnosis of aneurysm of the thoracic aorta having been readily made, medical treatment, as outlined below, was instituted. It consisted of absolute rest in bed in the semi-orthopneic position, attention to diet, and the administration of certain medicinal agents.

A modification of Tufnell's diet was used, as follows: Breakfast: a soft-boiled egg and two ounces of bread; dinner: three ounces of meat and two ounces of toasted bread; supper: milk toast. Between meals he was allowed four ounces of milk every three hours, but no other fluid. Later a small quantity of stewed fruit was added to the diet.

The medicinal treatment consisted chiefly in the use of tincture of aconite,

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mliij, every three hours, gradually reduced to mlij, t. i. d.; and a saturated solution of potassium iodide, mlv, thrice daily, increased mlj daily until the dose became mll, t. i. d., when the patient developed symptoms of iodism; it was now reduced to mlxxx, which amount was maintained during his entire stay in the hospital. Codeine sulphate, gr. ½, was administered by mouth when necessary to control the troublesome cough. He was also given magnesium sulphate in doses sufficient to keep the bowel movements in a fluid state.

On January 29, no improvement having occurred in his condition and there being now imminent danger of rupture, it was decided after consultation to resort to more radical measures, and accordingly one of us (J. P. Mann) inserted into the aneurysmal sac about ten yards of fine silver and copper wire. Almost immediately the expansile pulsation was markedly diminished and the tumor mass was less prominent. The wire could be distinctly felt, giving the impression of subcutaneous emphysema. About two weeks later an additional slight elevation above the second rib and to the right of the sternum was visible. This also gave an expansile pulsation. The second eminence slowly increased in size, and on February 13 Dr. Mann inserted several yards of fine silver wire. At this time the pulsation in the original mass was not apparent. In a few days the pulsations in the second elevation were less marked, and a considerable diminution in its size was noticed. On February 28 (within three weeks following the original operation) the entire upper surface of the primary tumor mass sloughed off, exposing the wire coiled up within the aneurysmal sac. A firm granulating surface was visible beneath the wire, which admirably fulfilled the purpose for which it was intended.

As the result of the formation of this granulation tissue and the progressive retraction of the sac, the wire was steadily protruded from the wound and small portions were removed each day until little remained. The coils of wire which had been passed through the sternal opening, however, were not removed. For a long time after the mass had disappeared there was a slight oozing of blood from the wound, and on June 16 a small hemorrhage occurred, about two ounces of blood being lost. On July 1 the patient first sat up. The wound now slowly healed by granulation. A slight pulsation over the second elevation was still present. The patient's strength gradually returned, and on July 17, the wound meanwhile having healed, he was discharged from the hospital.

Dr. Mann has made a supplemental report, as follows: "The aneurysm was first wired on January 29, 1907, at which time and continuously during the entire treatment of the patient careful precautions to insure asepsis were maintained. With a hollow needle of sufficient caliber to freely admit the passage of No. 26 silver wire the tumor was punctured near the base of the portion external to the sternum.

"About thirty feet of wire was passed through the needle, which was directed toward different areas of the sac, in order to better distribute its coil and also

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that the opening in the sternum might be blocked. The sternal aperture, as discovered by a radiograph, had a diameter of about three-fourths of an inch, and was located in the lower half of the gladiolus. Pulsation almost entirely subsided soon after the wire had been introduced.

"About two weeks subsequent to the first wiring—February 13, 1907—a small area of pulsation appeared in front of the sternum just above the original tumor. This second sac was filled with seven feet of wire, after which and up to date (January 15, 1908) there has been only feeble pulsation and no extension of the parts wired."

The patient has been seen at intervals of one to two months since he left the institution, and has continued symptom-free with the exception of slight dyspnea on active physical exertion. There are, however, certain physical signs still present, namely, very slight bulging over site of upper elevation, feeble pulsation (slightly expansile) over the same area, a small, abnormal field of dulness above and to the right of the normal cardiac dulness, and slight accentuation of the second aortic sound, but no bruit can be detected.

February 6, 1908, a small, shallow protuberance covering an area about the size of an ordinary coat button and of a dark-red color has recently appeared. This small, projecting mass exhibits an expansile pulsation. Finally, for about one year, the aneurysmal growth external to the sternum was apparently cured, and it is believed that the slight projection which has quite recently reappeared at the site of the original mass can be successfully wired.

An *x*-ray examination made this day shows no perceptible increase in the size of the saccular aneurysm situated to the right of the sternum when compared with the results obtained by the same method six months previously, The aneurysmal dilatation of the transverse portion of the arch, however. has extended itself to a slight extent, although it has given rise to neither symptoms nor physical signs.

March 10, 1908. The small protuberance, which is mentioned above as having appeared on February 6, 1908, has entirely disappeared, as the result of rest.

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THE X-RAY DIAGNOSIS OF ANEURYSM OF THE THORACIC AORTA.[1]

By HENRY K. PANCOAST, M.D.

Conservatively speaking, the *x*-ray examination in connection with aneurysms of the thoracic aorta may be considered as an exceedingly valuable and accurate method of observation, and should always be employed in addition to and as an adjunct to the clinical examination, but it should not be regarded as an entirely independent and strictly infallible means of diagnosis in this condition.

In regard to the choice of methods to be employed, those who have had most experience with the fluoroscope consider it the more satisfactory and the more reliable of the two in the long run. Aside from the question of the advantages and disadvantages in general of each method, the additional advantages which the fluoroscopic examination may possess in connection with aneurysms do not, from a personal point of view, seem to justify the risks entailed. Already some eight or nine individuals engaged in *x*-ray work have paid the penalty of their lives, and more are likely to follow their example, while many others are constantly reminded of their carelessness by the absence of fingers or hands, painful skin lesions, or permanent disfigurement. There is no method of employing the fluoroscope which is absolutely free from danger, and some, at least, of those who persist in its routine use will surely pay the penalty sooner or later. Under the circumstances, the subsequent remarks apply to the radiographic method of examination.

[1] Read February 5, 1908.

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There seems to be a tendency prevailing in some minds to regard the evidence of the radiograph for or against the presence of aneurysm as invariably conclusive in itself. Such views are not practical, and are certainly not those held by conservative radiographers. Undoubtedly in many instances aneurysms have been diagnosticated from the radiograph when the most careful clinical examinations failed to detect them. In a certain proportion of such instances, however, the clinical examinations failed to reveal what in reality did not exist. It is also true that the skiagraph has not infrequently failed to show evidence of an aneurysm that was undoubtedly present. In other words, the *x*-ray diagnosis of aneurysm is by no means to be regarded as an easy one to make, because it is often beset with difficulties and there are numerous sources of error associated with it. Nevertheless, the fact remains that the *x*-ray examination is one of the most valuable means of diagnosticating aneurysm at our command.

The question naturally arises as to whether some of the sources of error cannot be eliminated and the value of this method materially increased by lessening the likelihood of mistakes. Fortunately this question may be answered in the affirmative. Moreover, it is not unlikely that a radiographic substitute will soon be found for the fluoroscope, equally as reliable, if not more so, and free from the dangers associated with the use of the latter. It is too much to expect, however, that sources of error will be entirely eliminated. Primarily, there are two important factors that will be concerned in increasing the reliability of the radiographic examination of the thorax. They are:

1. Increased experience of both the radiographer and the clinician in studying and interpreting skiagraphs of the chest. Greater familiarity with the skiagraphic appearances of the normal aorta and certain anomalies, which are hardly to be considered pathological, of aneurysms, especially in those cases in which the diagnosis is certain or is confirmed later by autopsy, and of such pathological conditions as may simulate aneurysm, either clinically or in radiographic appearance, will undoubtedly tend to make the *x*-ray diagnosis of the latter condition more accurate and reliable

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2. More rapid radiographic exposures. The improvements which have enabled us to radiograph the chest while the patient holds his breath have already rendered the skiagraph much more accurate. Apparatus has recently been devised which makes it possible to skiagraph any thorax in a second, and that of the average individual in a fraction of a second. It is, therefore, now within the range of possibility to obtain satisfactory stereoscopic radiographs of the thorax in any case, and those who are familiar with stereoscopic radiographs will realize what this means.

Aneurysms of the aortic arch are especially adapted to the *x*-ray examination because of the nature of the surrounding structure. Lung tissue is easily penetrated by the rays, and any structure, normal or pathological, which is surrounded by it and differs from it in the slightest degree in density casts a perceptible shadow. Therefore the heart and great vessels are easily observed wherever other shadows do not obscure them. It is true that the shadow of the normal aorta is nearly or entirely obscured by that of the sternum and spine, but this is of no consequence, in view of the fact that any displacement or enlargement of the former casts a perceptible shadow beyond that of the latter. In perhaps the majority of individuals the shadow of the descending portion of the arch extends slightly to the left of the spine. This is not infrequently mistaken for aneurysm.

The important point, primarily, in the interpretation of the radiograph is to determine whether there is any abnormal projection of the shadow in the locality of the aortic arch beyond the normal limits. If the skiagraphic details are satisfactorily clear, and there is no such abnormality of the shadow, aneurysm of the arch *may be excluded.* If, however, there is any distinct departure from the normal size and outline of the shadow, the next point to determine is whether or not this is indicative of aneurysm or of some other condition. This is a question that cannot always be decided by the *x*-ray examination alone. It is here that sources of error become especially prevalent, and they are possible even in the best skiagraphs. The more complicated the shadows and the more doubtful the skiagraph the greater the possibility

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there is of error. When there is the least degree of doubt, an *absolute* x-ray diagnosis of aneurysm should neither be given nor expected. The radiographer should embody his findings in a statement as to the most likely condition to which his examination points.

From the radiographic standpoint we may recognize two classes of aneurysms of the arch primarily, namely, the diffuse and the localized or more or less sacculated.

DIFFUSE. In the diffuse aneurysm, involving more or less of the entire arch, the shadow extends uniformly for a considerable distance beyond the normal limits on one side or the other, but usually in both directions. After first determining beyond all doubt that this is the shadow of the aorta, the next question to decide is whether or not it is due to an aneurysm or to a dilatable aorta. Diffuse aneurysms, when not marked, are often exceedingly difficult to differentiate with certainty from the latter condition. One of the errors most frequently made is to mistake a dilatable aorta for an aneurysm. There is one essential point in which these two conditions differ, and by which they are to be differentiated. The enlarged shadow of a dilatable aorta nearly or entirely disappears between pulsations, while that of an aneurysm does not. In the radiograph two distinct shadows are to be distinguished in the former condition. One is larger and represents the extreme degree of dilatation of the vessel. The other is smaller and represents the walls of the vessel during diastole.

LOCALIZED. In the more localized, or more or less sacculated variety, the enlarged shadow is to be seen projecting either to the right or left of the normal one. Such aneurysms may involve any one or two portions of the arch, or the descending thoracic aorta.

1. *Ascending Portion.* Aneurysms involving this portion will cast a shadow to the right side, above that of the heart. Better details may be obtained with the rad ograph made from the front, as this portion of the arch lies more anterior than the others. In thin subjects it may be possible to obtain a satisfactory lateral view, in which the shadow may be seen filling the anterior clear space between the heart and the front wall of the chest.

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2. *Transverse Portion.* In small aneurysms involving this portion of the arch the greater part of the abnormal shadow may be directly upward, in which case it is most difficult or even impossible to detect, for the same reasons that the normal shadow is obscured. The stouter the subject the more likelihood there is of such a lesion escaping detection. Lateral views in such cases would be of considerable assistance, but unfortunately they are most unsatisfactory in this locality. Detection depends largely upon the clearness of the radiographic details. If the shadow extends laterally, however, as it usually does, and especially to the left side, its detection is much more certain. If the aneurysm is large the diagnosis is usually not difficult. A radiographic feature upon which considerable importance has been placed is the change in the position of the heart often observed in these cases. It is said to lie more horizontal than normal, as though the base were pushed downward and to the right. The diagnostic significance of this appearance has probably been exaggerated, as such an apparent abnormal position of the heart does not necessarily have to be associated with aneurysm.

3. *Descending Portion.* In aneurysms of this portion of the arch the abnormal shadow is of course observed extending to the left side, in the first interspace and behind the first rib. A dilated or hypertrophied left heart is likely to be confusing because its shadow overlies that of the aorta and extends upward to the arch. Under such circumstances, unless the aortic shadow can be differentiated from that of the heart, which is not always possible in stout subjects, the interpretation of the plate requires the greatest amount of care and judgment.

4. *Descending Thoracic Aorta.* Aneurysms of this portion of the aorta cast an abnormal shadow to either side, but it is particularly noticeable to the left side. If we depend entirely upon the anteroposterior view the aortic shadow must be differentiated from that of the heart, or otherwise an aneurysm cannot be detected. An aneurysmal shadow is of course more dense than that of the normal aorta, and aneurysms of this region can usually be readily detected; but in stout subjects they are not always

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easy to demonstrate. Their usual locality, as observed by radiographers, seems to be just above the point where the vessel passes through the diaphragm, whence the swelling extends for a variable distance upward. In the three cases of this kind that I have observed the shadow has been spindle-shaped, representing probably an early stage. It is possible that two of these were cases of dilatable aorta and not of aneurysm. In advanced stages the shadow probably becomes more rounded. Baetjer observed one case of aneurysm in this locality, with the pulsating shadow rounded and about the size of an orange. The heart was pushed upward and outward. It is possible that some supposed abdominal aneurysms are in reality lesions of this portion of the aorta. Such was the case in one of the three to which reference has just been made. A satisfactory lateral view, if obtainable, should show these aneurysms to the best advantage, the swelling filling up the retrocardiac clear space behind and below the heart.

Differential Diagnosis. 1. *Mediastinal Tumors.* It is not difficult to distinguish between an aneurysm and a mediastinal growth, provided the shadow of the aorta can be seen. If there is but one uniform shadow, then the diagnosis may be extremely difficult, and the radiographer, in order to interpret the plate with the greatest possible degree of accuracy, must depend upon the clinical data equally as much as the clinician expects to depend upon the *x*-ray findings. The clinical history of the case is often most important, of course. The shadow of a tumor which entirely obscures that of the aorta is apt to be denser than that of any aneurysm would be. It is said that the edges of a growth are apt to appear more or less irregular, and not clean cut, but this is certainly not always the case. With the fluoroscope, the absence of pulsation strongly points to tumor, but a tumor may move by transmitted pulsation, and this movement may be mistaken for the expansile pulsation of an aneurysm. Ordinarily the *x*-ray diagnosis between these two conditions is reasonably certain, but it is not so in all cases.

2. *Enlarged Mediastinal and Bronchial Lymph Glands.* The shadows of these are apt to appear prominently at a lower level

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than the arch and around the roots of the lungs. As a rule, they fade off into small scattered shadows, and all of them are relatively very dense. If the aortic shadow is distinguishable there is no difficulty in the diagnosis.

3. *Localized Empyema.* This needs hardly more than mere mention as a condition which, under certain circumstances, might possibly be confused with aneurysm.

4. *Thickened Pleura.* The same remarks are applicable to this condition.

5. *Displacement of the Aorta.* This condition might be associated with scoliosis or marked deformity resulting from spinal caries, but it is hardly likely to lead to a diagnosis of aneurysm.

6. *Tortuosity and Other Variations of the Aorta.* There is considerable variation in the width of the shadow of the normal aortic arch in healthy individuals. Although it is usually uniform and extends little if any distance to either side of the shadow of the spine, it not infrequently projects beyond this limit, either to one or both sides. In quite a large percentage of normal individuals the descending portion of the arch projects considerably to the left, and then turns sharply toward the spine, making a small, but decidedly prominent, shadow which may easily be, and probably often is, mistaken for a small aneurysm. Familiarity with this appearance should be a sufficient safeguard against such a mistake.

7. *Dilatable Aorta.* This condition has been discussed in connection with diffuse aneurysm.

In addition to these conditions from which aneurysm must be differentiated, and for which aneurysm may be mistaken, there are certain others which may interfere with the *x*-ray diagnosis of aneurysm. Chief among these are pleural effusions. Simple effusions are not infrequently associated, directly or indirectly, with thoracic aneurysms. Any fluid filling the pleural cavities may obscure the heart and aorta completely. In any case in which aneurysm is suspected, and a pleural effusion is found, the fluid should be removed and the case again examined, if there is any likelihood of the effusion having obscured an aneurysm.

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CONCLUSIONS. 1. The appearance of normal shadows in the region of the thoracic aorta is the most reliable evidence against the presence of aneurysm.

2. A definite *x*-ray diagnosis should not be rendered unless the skiagraph is satisfactorily clear in its details. There should be no hesitation in making two or more examinations if necessary in order to obtain satisfactory results.

3. Any aneurysm will cast a shadow. Failure to demonstrate it may arise from the presence of conditions which obscure it, poor negatives, faulty technique, or failure or inability to interpret the radiograph correctly.

4. Other conditions, such as newgrowths, etc., may cast shadows simulating aneurysms, and it may be difficult or even impossible to determine the correct diagnosis, but it can be determined correctly in the large majority of cases.

5. The *x*-ray diagnosis may often be made at a much earlier stage than a clinical diagnosis, and at a time when the discovery of the condition is of the greatest importance from the therapeutic standpoint. It is not necessary to wait for definite clinical signs if they are not yet present.

6. In cases in which the clinical diagnosis is more or less uncertain the evidence of the *x*-ray examination is of great value in the large majority of instances.

7. Even when the clinical diagnosis is apparently certain, the *x*-ray examination is of value, not only as confirmatory evidence, but also to demonstrate accurately the size and position of the aneurysm, and the degree of pulsation. Moreover, it is a safeguard against possible errors in the clinical diagnosis.

8. There is reason to believe that increased experience and improvements in methods and in apparatus will render the *x*-ray examination for aneurysm still more accurate in the near future.

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THE PATHOGENESIS OF ANEURYSM OF THE AORTA.[1]

By JOSEPH McFARLAND, M.D.

I SHALL present the pathogenesis of aneurysm only in a very elementary fashion. We are still uncertain as to the etiology of aneurysm, and usually sum up the factors concerned as two: the blood pressure and a morbid condition of the vessel wall. When these two factors coöperate the explanation seems simple. When either one fails, the explanation is more difficult. The force of the blood pressure may become great and the aorta be much diseased, yet no aneurysm occur. On the other hand, the blood pressure may be increased and the vessel be normal, and yet aneurysm develop. The specimen with which I begin this demonstration is a very remarkable one, and teaches a lesson of great importance. The history of the patient is as follows: He was a man about thirty-eight years of age, who came into the Philadelphia Hospital complaining of great pain in the precordial region. The resident physician found something wrong with the heart, the nature of which he was unable to make out. Seeing that the cardiac action was hurried, he concluded that the man needed digitalis. The patient consequently received a few doses of this remedy, was suddenly seized with great pain, rose and knelt beside the bed, pressing his pericardial region against it, declaring it to be impossible for him to move on account of the pain. A few hours afterward he died. No visiting physician saw the man. When the case came to the autopsy table the pericardium was found distended with blood and quite a rupture in the wall of the aorta immediately above the aortic valve. This was a transverse

[1] Exhibition of specimens, February 5, 1908.

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laceration, very clean cut. The aorta showed no signs of disease, or, at least, such insignificant signs and so remote from the seat of laceration that the entry on the record was that "the aorta showed no signs of disease." We have, then, in this case what seems to be a perfectly normal aorta undergoing spontaneous rupture on account of abnormally great blood pressure. You will perhaps recall that some time ago Dr. Daland reported a case, to which Dr. Osler makes reference in his text-book, of an aneurysm that had its beginning in just such a transverse laceration of the aorta, in just about the same position. If this laceration had not extended so as to permit fatal hemorrhage there would in time have formed an aneurysm. This then exemplifies what I said, that abnormal blood pressure acting upon a supposedly normal vessel may result in aneurysm.

Here is an aorta every bit of the surface of which is seamed and scarred and ulcerated by arteriosclerosis, yet without any aneurysmal dilatation which exemplifies that blood pressure acting upon badly diseased vessels may produce no aneurysm.

The morbid conditions that predispose to aneurysm are not definitely known. Syphilitic lesions are supposed to be among the chief predisposing causes, because of their destructive nature, but most of us conclude from what we see of arteriosclerosis that not only syphilitic lesions but others may do the same. There is no sharp point at which dilatation of the aorta ends and aneurysmal dilatations begin.

In arteriosclerosis of the aorta it is quite a common thing to find a rigid, greatly dilated aorta, these dilatations passing through all grades of enlargement until the pouch formed at the arch of the aorta is as large as one's fist and it becomes difficult to say whether the condition is a fusiform aneurysm or simply a dilatation of the aorta.

If in the formation of the aneurysm all the coats of the blood-vessel are preserved, we have true aneurysm. If there is rupture of the coats, which almost invariably occurs, we have false aneurysm. Many have endeavored to determine which of the coats yielded first. This, it seems to me, makes no difference, nor does it seem

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to me to be either of practical or scientific interest to pursue this differentiation. In most cases it is difficult or impossible to make.

What does interest us as clinicians is the final outcome of the case. It almost always happens that if the patient lives long enough the dilatation will result in such thinning of the wall that rupture and fatal hemorrhage will take place.

In regard to treatment the ambition of the clinician seems to be to accelerate that which usually takes place spontaneously in nature, that is, the clotting of the blood. We sometimes find currant-jelly clots, sometimes laminated clots surrounding a mass of currant-jelly clots. Sometimes we find aneurysms in which the entire interior is filled with a mass of coagulum that seems to be very old. This is the thrombosis supposed to be so desirable in promoting the cure of the affection. In the cases in which the formation of firm thrombi occurs it does not appear to me that the condition of the patient is materially improved. Unless it should so happen that the thrombotic mass enclosed in the aneurysm should obstruct the orifice by which the blood enters it, or should on all sides form a firm adhesion to the wall so that blood could no longer pass about it, the patient is no better off because he has the thrombi in the aneurysm than if he has not. The densest thrombi can be formed in the aneurysmal sac and yet blood keep constantly passing between the mass and the aneurysmal wall, and so long as the least quantity of blood enters under pressure and separates the thrombi from the aneurysmal wall the aneurysm will continue to grow in size.

Multiple aneurysms we frequently see in different stages of formation, some in which there are few clots and others in which there are very many. So far as we can make out the general tendency of all aneurysms seems to be in the direction of increase, and though there are cases on record in which aneurysms have spontaneously recovered, I have yet to see the first aneurysm in which there was clear evidence that the growth of the aneurysm had ceased.

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THE WIRING OPERATION IN THE TREATMENT OF ANEURYSM OF THE AORTA.[1]

By HOBART AMORY HARE, M.D.

There are several points that I wish to bring before the College, not so much in the description of the operation itself, as in regard to the remarkable results which follow.

The first and most definite result which followed the wiring operation in every case in which I have performed it, or seen it performed, is the remarkable diminution in pain which takes place, the patient, usually before the operation is finished, stating that pain is very greatly decreased, whereas one would suppose that the introduction of a large needle and a number of feet of wire would produce a degree of traumatism which for the time being would cause an exacerbation of pain.

A second noteworthy point is the diminution in the patient's dyspnea, or rather in the symptoms of pressure before the operation is finished. In the case of a man seen at the Hotel Walton with Dr. Musser, the suffering was so great that the patient said, "I want to say to you that whatever your decision is, if this thing is not done, I intend to kill myself, for I will not spend another twenty-four hours suffering this agony both as to the difficulty in breathing and as to pain." Before the electricity had been passing through the tumor for fifteen minutes the patient expressed himself as wonderfully relieved.

The next point of interest is the fact that, so far as I know, in some sixty-odd operations of this character performed and recorded,

[1] Read February 5, 1908.

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there is not a single instance in which an accident has occurred during the operation. In no instance has there been rupture of the sac or the sweeping off of a loose clot producing a secondary lesion. In the case of Dr. Musser's to which I referred, a very interesting coincidence occurred. The operation was to have been performed at 12 M. on Wednesday. A few hours before, the patient became partially hemiplegic and aphasic. The condition was undoubtedly due to embolism, because later on it entirely disappeared. If the embolism had developed after the operation it would have been attributed thereto.

Another point is that the operation is never permanently curative, except in those rare instances in which, as the result of trauma, one single portion of the aorta is seriously damaged. In the vast majority of cases in which aneurysm occurs it takes place as a manifestation of general vascular disease, and the history of all the cases I have operated on, and of all I have seen reported, has been that sooner or later after the wiring the adjacent tissues of the bloodvessel have given way. The proposition is similar to that of mending a piece of rotten hose: when it is mended in one place pressure causes it to break in another.

There are many cases on record in which life has been prolonged for considerable periods of time in comfort and usefulness to the patient by the performance of this operation. A patient of the late Dr. D. D. Stewart lived for a little over three years following such an operation, and died of alcoholic pneumonia and not because of the aneurysm.

In the case operated upon for Dr. Musser, two years ago in April, although the man was in such distress from dyspnea, he recovered sufficiently to return to South Carolina. Although he was on his back for a period of six months after the operation he constantly attended to business. Last October he passed through this city on his way to New York, where he actively engaged in business. At that time Dr. Newcomet radiographed him again, and the picture showed excellent effects. The man returned here about six weeks ago, and was seized with an attack of la grippe.

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He developed an acute pulmonary edema, either as the result of la grippe or of the aneurysm, and died in the course of a week or ten days. An autopsy was not obtained.

The case operated on with Dr. Fussell was the only one in the descending aorta and going through the back that I have wired. This patient lived four months and died from pressure and exhaustion.

Another point is the exercise of great care that the needle is not pushed at the time it is inserted, or later, by inadvertence, so far into the aneurysmal sac that the shoulder of the needle comes in contact with the skin. The shoulder of the needle is never insulated, and if the greatest precaution is not taken an electrolytic burn will result. A slough takes place which decreases the strength of the aneurysmal wall, and in one case which I operated upon death came because of this accident. I was called out of the operating amphitheatre to answer the telephone when the operation was about half over. The assistant pushed the needle in too far and burnt the skin. The man returned to his home in the South, but ultimately died by losing an enormous worm-like mass of clot three or four times a day through this opening in the skin. The aneurysm had developed, not as the result of arterial disease but of an injury sustained by being thrown from his horse, striking on the pommel of the saddle.

In one instance only have I wired the innominate artery. The man died four months afterward as the result of pressure symptoms and exhaustion.

I have used gold wire in all my cases. I am not in a position to criticise silver, but I imagine that it has one advantage and one disadvantage. The advantage is that it is a little easier to handle, and the most delicate part of this operation is the introduction. The soft gold wire is very apt to buckle. On the other hand, when the wire gets into the mass, I should imagine that the gold wire would better adjust itself to the uneven surfaces of the sac than would silver. Usually from 70 to 80 milliampères are employed and the operation continued for not less than from fifty minutes to an

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hour. The hardening of the clot does not take place for several days or weeks. In the last case seen by me, in which I assisted Dr. J. Chalmers Da Costa, the man expressed his relief within ten minutes after the electrical current was passed through the wire. In the operation which I performed on the innominate aneurysm, although the autopsy was made by a most competent, skilful, and experienced pathologist, and although no less than 18 feet of gold wire was inserted, not a trace of gold wire could be found in any portion of the patient's body!

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SACCULATED ANEURYSM OF THE ARCH OF THE AORTA RUPTURED INTO THE SUPERIOR VENA CAVA, WITHOUT SYMPTOMS OF VENOUS OBSTRUCTION.[1]

By JOSEPH SAILER, M.D.

Perforation of the veins by aneurysms is very rare, and in all the cases with which I am familiar perforation has been associated with visible signs. Speculating upon the cause of the absence of these visible signs, it is not possible to exclude some exceedingly rare anomaly which might permit obstruction to take place in the descending cava and at the same time permit an easy collateral circulation to be established. I know of nothing in anatomy which would justify such a supposition. The reflux of blood through the inferior cava did not seem to make it any easier to understand why the obstruction did not also occur in the superior cava. The only plausible explanation is a purely hypothetical one, and does not satisfactorily account for the condition. It is that in some way in this large sacculated aneurysm the pressure of the blood in the sac was not as great as it must have been in the rest of the arterial system. In other words, the pressure in the sac at the site of the opening must have been approximately equal to the pressure of blood in the veins. It is difficult to understand how this could occur, but the appearance of the specimen suggests this possibility. The current of blood may have been sufficient in part either to diminish the pressure of the sac, or set up an eddy of blood in the sac, preventing the blood from being forced directly through the cavity.

[1] Exhibition of the specimen, February 5, 1908.

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DISCUSSION ON THE PAPERS OF DRS. ANDERS, HARE, McFARLAND, AND SAILER.

DR. JAMES P. MANN: I have had the surgical treatment of the case since the original examination in January, 1907. The tumor, as Dr. Anders has said, was in a condition which foreboded rupture in a short time, and in order to save the patient, resort to other than medical interference was necessary. I determined to wire the aneurysm, and this was done with silver wire. Upon one occasion I took silver wire from a bone, where it had been for at least fifteen years, and it was as bright on removal as when put in. With the head of the needle bevelled the wire can be introduced much more easily. No. 26 silver wire was used, as it was thought to give sufficient rigidity and yet coil easily after entering the aneurysm. A needle just sufficiently large to take the wire easily is better than a large needle, and the wire is directed to different parts of the sac in order to fill the aneurysm. In this case, after the sac began to slough, we found that the wire had not only passed into the external sac, but through the opening in the sternum. This was sufficient to cause blood clot; otherwise there would have been fatal hemorrhage. The patient did well until a few days ago, when there was observed, at the site of the original aneurysm, a reforming tumor.

DR. ROBERT N. WILLSON, JR.: I have with me a large aneurysm of the descending aorta, containing fifteen feet of gold wire coiled in and around a clot. I have operated upon three cases of aneurysm. Two are now living and one died within a week or two after wiring. The patient was more comfortable before wiring than afterward. This was directly opposite in its results from another case which I wired four years ago, and which is still living. This was a case of aneurysm of the arch which had been wired twice before. The man was dyspneic and edematous and about to die, as we thought. I saw him for some time in my office after he left the hospital. Fifteen feet of wire were introduced and the current raised to 20 milliampères, and in this case we burnt off the wire while using the current. I felt and still feel that we probably destroyed the whole of the wire in the sac of the aneurysm. The *x*-rays revealed no trace of the wire in the plate, and it was no doubt destroyed by the high current. Of the three aneurysms that I have wired, therefore, the results in two were very favorable, and in one we seemed to do more harm than good.

DR. JOSEPH McFARLAND: I think Dr. Willson is right in regard to the aneurysm in question. At first I thought it arose from the iliac artery, but now I believe it arises from the abdominal aorta, just below the celiac axis, and involves the iliac and femoral by extension. I should like to ask Dr. Hare whether there is any known duration of aneurysm by which one can judge that the life of the patient is definitely prolonged by the operation of wiring.

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Dr. William S. Newcomet (exhibition of plates): Almost all of the pictures were confirmed with the fluoroscope. In regard to the danger of this method, if the fluoroscope is used with care, when the *x*-ray tube is protected, and the operator is not exposed to the direct rays, it is very slight. Dr. Williams, of Boston, has been employing this method of examination ever since the *x*-rays were brought into use, with no untoward result, while many others, who have not been so careful and yet not used the fluoroscope, have been severely burned. The fluoroscopic examination does not give detail, but in such cases as aneurysm it is of marked value, as you are able to see the pulsation of the mass not only in one plane, but in several. This is a decided aid when such operations are contemplated as have been just described by Dr. Hare.

Dr. M. Howard Fussell: Some years ago I went over the literature of the subject on rupture of aneurysm into the superior cava. At that time I collected thirty-six cases of such an accident, including cases reported by Pepper and Griffith in 1890. In all of these cases the symptoms of sudden cyanosis, edema, and dilatation of the veins of the face, neck, arms, and chest were present. In about one-half of the cases there was a peculiarly continued high-pitched murmur. The edema and cyanosis always came on suddenly.

There is one case on record in which these sudden symptoms of cyanosis, etc., occurred in aneurysm of the arch of the aorta, but very careful examination of the specimen postmortem failed to show the slightest connection between the aneurysmal sac and of the vena cava. It was supposed that the pressure was caused by a sudden dilatation of the aneurysmal sac.

The specimen which I present this evening was procured from a patient at St. Mary's Hospital. The specimen shows a very small aneurysm of the arch of the aorta; inflammation had taken place about the aneurysm, and the inflammatory material had connected it with the vena cava. The rupture of the aneurysm itself had occurred considerable time before the symptoms, but the rupture into the vena cava occurred suddenly with the occurrence of the symptoms. The communication with the vena cava is as large as a three-cent silver piece. The man lived about three weeks after the beginning of the symptoms.

Dr. J. Alison Scott: I should like to give my experience, especially in regard to the wiring of aneurysms. Dr. Hare has spoken of not hearing of any untoward results. A few years ago I had under my care at the Polyclinic Hospital a man with aneurysm on the left side of the neck. It was subsequently wired by Dr. Daland. Within twelve hours after wiring the man developed multiple emboli, was generally paralyzed, and died within two or three days.

I have under my care at the present time a man who was wired on July 2d. He then had an aneurysm of the innominata the size of a small lemon.

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He was wired, and was better for about three months. He now has an aneurysm the size of a cantaloupe spread out all over the entire right side of the neck, extending across the trachea and pushing it to the left side. The man is in bad condition. The aneurysm was wired during Dr. Le Conte's service by someone who has done a great deal of this work in the city. In another case, an aneurysm of the ascending aorta increased three inches in two days and then diminished much in size in five days. This evidently was a false aneurysmal sac; otherwise I think the variation in size would not have been nearly so great in such a short time.

In another case, a man with aortic regurgitation, there was a great difference in the two pulses. There was constant and severe pain. The man subsequently died, and the autopsy showed a remarkable condition in the aorta. He had a large, much dilated aorta. The subclavian artery was almost entirely occluded with an atheromatous plaque.

I do not understand why wiring of an aneurysm should cause sudden and remarkable decrease of pain. The pain is due to pressure from erosion of bone by increase of the sac or due to pressure on nerves. It must be the electricity which causes the sudden diminution of pain. It cannot be due to the sudden ceasing of the pressure, unless the sac diminishes tremendously and very rapidly in size. The question of Dr. McFarland becomes very important. We do not know the ordinary duration of aneurysm. I have seen one woman live eighteen years with aneurysm which has been demonstrated for years and years. She had been making her living for a number of years in this way, and died about a year ago.

Dr. John B. Roberts: It may be interesting to recall that the first work of this kind was done in this country, perhaps in the world, in the Pennsylvania Hospital, in 1873, by Dr. R. J. Levis, who introduced horsehair into a subclavian aneurysm by pushing it through a small cannula. The man appeared better for a few days and then died. There was no such thing as asepsis in American surgery in those days.

Dr. Arthur V. Meigs: I recall very distinctly the case Dr. Roberts refers to, as I assisted Dr. Longstreth, the pathologist to the hospital, in making the postmortem. There was found to be about as much of the horsehair in the lung as in the aneurysm.

Dr. M. Howard Fussell: I want to mention a case operated upon for ovarian cyst, and in which at autopsy there was found an aneurysm entirely cured.

Dr. Mann: Dr. Anders requested me to say that this operation was not done with the idea of a permanent cure, but that it did relieve this man of his symptoms and the physical signs and enable him to go back to his work and prolong his life. How much longer he will live of course we shall find out later.

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I selected silver wire because it coils itself better and passes through the needle with less liability of kinking. The kinking can be prevented in a measure by putting it upon a spool.

Electricity was not used in this case because almost from the time the wiring was finished the pulsation had ceased in the tumor. It was, therefore, not considered necessary, and not advisable, owing to the condition of the skin, which was necrotic, with hemorrhage almost imminent. If we had not wired this case the man would have died in a few days.

DR. SAILER: I had the same impression that Dr. Fussell expressed, that my case was absolutely unique. I have been unable to find reference to any other case, although I have not made as thorough study of the literature as I could wish. I agree with Dr. Fussell as to the difficulty of distinguishing between true and false aneurysm. Excepting in slight fusiform aneurysm, the differential diagnosis is exceedingly difficult. Some years ago Dr. Pfahler and I undertook a rather elaborate series of investigations with the *x*-rays. We studied the plates carefully, and it seemed to me that there were a good many points about these plates, and particularly the diagnosis of aneurysm from *x*-ray plates, which are of a good deal of interest. I think particularly the changes that take place in the atheromatous aorta, in which the vessel is more or less permanently dilated and some of the elasticity of the wall is lost, are of interest. We studied nearly all these cases with the fluoroscope as well as with the photographic plate. Then the fluoroscope was not regarded with the terror that it is at present, and we watched our cases for considerable periods of time. I do not think that in any of those cases it could be fairly said that the aorta returns even approximately to its normal size during diastole of the heart. I do not think the fluoroscope enables us to make a differential diagnosis. Other points of differential diagnosis, however, which we attempted to explain in a paper, were based upon those cases.

DR. HARE: I presume we shall never have very accurate statistics in regard to the duration of life in aneurysm, because every case is a rule unto itself. I do know, however, that when a man has an aneurysmal sac, with the skin about it blue and rupture imminent, that wiring has caused the pain to cease and the man has lived for many months. I think, therefore, that the operation has something to do with the prolongation of life. In the case of Dr. Musser, the plates of which Dr. Newcomet has shown, the man suffered with pain and dyspnea to an extraordinary degree, was bedridden, and yet he was able to go back to the South, and I understand from Dr. Musser that he told him he was able, by reason of the number of months of his improved health, to save himself many thousands of dollars which he would have otherwise lost.

Regarding the case quoted by Dr. Scott, I should like to mention that in one of the cases in which I operated I upbraided the electrician for attempting to touch the needle, when he became so upset that when I told him to attach

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the wires to his battery I found him with the positive pole wrongly attached and the aneurysm connected with the negative pole, thus producing an alkaline reaction and dissolution instead of acid reaction and coagulation. Perhaps in Dr. Scott's case some mistake like that occurred, and the old clot which existed before operation was dissolved by negative electrolysis and multiple emboli produced.

As to decrease in pain. Pain is a thing we cannot measure, but if a man who has been suffering extreme pain, and says that he is much relieved, we have got to believe that he is relieved. How much he is relieved perhaps we cannot tell. It is a well-known fact that electricity has in some instances a sedative effect, and, in addition, I would suppose that pain is decreased by the diminution in pressure because within ten or fifteen minutes after the passing of the electricity the excessive pulsation in the mass ceases and there is not that irresistible heaving impulse.

I have always used the fluoroscope with extraordinarily satisfactory results. At every stroke of the heart there was beautiful expansile pulsation in the mass, which we could see with perfect clearness. A skiagraph, which gives a fixed view, will not give any idea of the degree of excursion and direction of the pulsations.

As to the drug treatment of aneurysm, I have never seen any drug, barring aconite and veratrum viride, which would diminish the force of blood. I have never seen any drug produce the slightest effect upon aneurysm, even in those so-called syphilitic aneurysms in which iodide of potassium is thought to do good. I believe there is no more reason for supposing that the pathological processes which exist in aneurysm occurring in parasyphilitic affections are benefited by iodide of potassium than there is reason for believing that the late lesions of syphilis are affected materially by iodide of potassium. I think that the lesions are so irremediable that, so far as the aneurysm is concerned, the iodide of potassium is of little direct benefit.

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THE BACTERIOLOGY OF THE PUERPERAL UTERUS.[1]

By WILLIAM R. NICHOLSON, M.D.,
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AND

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(From the Maternity Hospital and the William Pepper Laboratory of Clinical Medicine of the University of Pennsylvania.)

THE germ content of the uterus has been controversial ground for years. The question has been investigated by many since the time of Doederlein with the most varied results, to be explained, in our opinion, upon the ground of errors in technique. Probably the earliest investigator in this field was Myerhofer, who, in 1865, employed suction by means of a glass tube. He was always able to demonstrate cocci and bacilli if the case were febrile, but he did not find them in afebrile cases. Doederlein, in 1887, reported a series of 30 cases with normal temperature. In 27 of these the lochia was sterile, while in 3 bacilli were present, streptococci being identified once. He drew the conclusions from his study: (1) That in normal cases the uterine lochia is sterile; (2) that microörganisms are almost always present in severe cases of puerperal infection; (3) that infection is not always due to lack of care on the part of the physician. Von Ott and Czerniewski, in 1888, arrived at the same conclusions, but the latter held that the strep-

[1] Read April 1, 1908.

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tococcus might be present in the lochia of normal cases in rare instances without symptoms.

In contradistinction to the above, we wish to call attention to the statistics contributed by Stolz, who found streptococci in the vaginal secretion of three normal pregnant women, while in a series of women with abnormal vaginal secretion during pregnancy streptococci were present in several instances. In studying the puerperal uterus, he found that on the ninth day, in a series of 156 cases examined during labor, with aseptic precautions, there were 55 positive and 101 negative results, streptococci being found in 11 per cent. of the positive cases.

Neither the duration of the labor, the presence of laceration, the number of examinations during labor, or the operations performed to effect delivery seemed to alter the result. Retention of secundines did, however, increase the bacterial content. In 54 women who had not been examined in labor, and in whom the puerperium was normal, he found that in 10, cultures gave streptococci on the ninth day. In a series of over a dozen women in whom cultures were taken on the third, fourth, fifth, sixth, seventh, and eighth day, in order to determine the time of ascendancy of the germs, it was found that early in the puerperium there might be non-pathogenic organisms in the lochia with absence of any signs of infection; that the puerperium from the third day on did not differ in germ content from the later period (ninth day); that the content on the ninth day was less on the average than earlier. Therefore, he believes that in the latter days of the puerperium the uterus cleanses itself from organisms in a large proportion of cases. As his cases were not selected, there occurred a series of 18 in which, one or more days after the culture, there was a development of fever. In this febrile series, with the exception of 5 cases, there had been found a growth in culture before the appearance of the fever, and in half of the positive findings streptococci were demonstrated. In cases in which, on the earliest culture, streptococci were present, there cannot be a question of having caused the fever by infection at the time of culture, accord-

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ing to the opinion of the author. Doederlein and Franz also conclude that in cases of streptococcic vaginal lochia the taking of a culture is not a harmless experiment, but that the infection is sometimes to be laid at the door of the culture. Doederlein declared that the initial period of freedom from symptoms before the taking of the culture could be explained by the incubation of the organisms, but Stolz denies this. However, he does not deny that the presence of streptococci in the cavum uteri may be the cause of the development of fever, without other reason than the trauma necessitated by the culture. He makes the point that the danger is much less if the culture be taken late in the puerperium, and states that he has never seen any difficulty if he has not cultured in the early days. In studying the examination of the lochia and vaginal secretions on the fourth day, he used a series of 75 cases for the uterine lochia and 65 cases for the vaginal secretion. He found that the uterine lochia was positive in 80 per cent., and that in 36.9 per cent. streptococci were present, while the vaginal secretion in 27 per cent. gave streptococci. Both these series were entirely normal cases. He concludes as follows in regard to the findings of the fourth day: (1) That the vagina always contains organisms, and the uterus does so in a large proportion of cases; (2) that the presence of streptococci in either situation is possible without symptoms; (3) that the uterus contains fewer germs in unexamined cases than in those examined, but streptococci are to be found in both uterus and vagina in some unexamined cases; (4) that examination with sterile hands during labor has no apparent influence upon the germ content; (5) that aseptic glove operations have no distinct influence; (6) that no actual increase in germ content appears to be dependent upon the duration of labor, but that streptococci are relatively increased.

Finally, we would quote rather extensively from the paper of Little, which appeared while this paper was in preparation. Little came to the following conclusions: (1) Cultures taken immediately after labor are sterile in 92 per cent., or in 96 per cent. if gonorrheal cases be included; bacteria may be present excep-

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tionally, even in the absence of vaginal examinations during labor, and the puerperium may be normal; the bactericidal power of the blood serum may be the cause of a certain number of negative results. (In 10 of his 50 cases it was necessary to introduce the hand into the uterus, and it is impossible to believe that no organisms were carried into the cavity. Of course, the downward flow after labor may also be a reason for negative findings in these cases.) Upon the third day he found sterility present in 62.5 per cent. of 40 afebrile cases, as compared with 40 per cent. sterility in 10 cases in which the temperature reached 100.6° or higher. (2) That the inclusion of cases in which gonococci were demonstrated will increase these averages to 85 per cent. and 50 per cent., respectively. (The author calls attention to the fact that he has included in his febrile series cases in which the temperature rise was obviously not due to any intrauterine cause, and the lochia, being sterile in these cases, or at least in some of them, a decrease in the percentage of the negative results is caused in the afebrile series and of the positive results in the febrile.) (3) That the finding of bacteria in the cervical canal at the completion of labor does not necessarily imply their presence later in the puerperium. (In one instance, in which there was a flaw in the tube, the author found a positive result in the first culture, but a negative one the third day, and admits that this was due to contamination with cervical secretion.)

Conclusions based on the cultures made on the seventh day postpartum were as follows: (1) Absolute sterility in 50 per cent. of the afebrile cases, as compared with 20 per cent. of the febrile. (2) Inclusion of gonococci makes these averages 80 per cent. and 50 per cent., respectively. (3) Bacterial findings differ more markedly according as the case is febrile or afebrile than by clinical characteristics. (In 5 of this last series positive results were obtained in cases which had been sterile at both previous cultures. From 3 of these 5 cases a diplococcus was obtained which resembled, and was probably identical with, the diplococcoid forms so frequently observed in the cervix and vagina, and the author thinks

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it may have been due to contamination, since he admits that it is impossible to get an absolutely trustworthy result by his method at this stage. In the 2 remaining cases the organisms were gonococci.) It is to be noted that in 20 cases showing bacteria on the seventh day of the puerperium the gonococcus was the organism in 12, and, further, that only 2 of the 10 febrile cases had sterile lochia on the seventh day; the streptococcus found in one instance on the third day had disappeared on the seventh day. (4) The uterus has the power of self-cleansing, and so the presence of organisms on any day of the puerperium does not mean their continued presence, and the author believes that the results given by Stolz, who found fewer organisms late in the puerperium than at an earlier period, are to be thus explained.

The gonococcal findings in his complete series of cases were demonstrated in 2.5 per cent., 25 per cent., and 30 per cent., respectively, on the first, third, and seventh days. His general conclusions as regards the effect of interference during labor are as follows: (1) Laceration of the cervix and the perineum have no bearing upon the germ content of the lochia; and (2) vaginal examinations have but slight effect (4 out of his 6 afebrile cases not examined during labor showed bacteria in the lochia, Bacillus coli communis being found once and the gonococcus three times; while 1 afebrile case was examined six times in labor and no organisms were ever found in the lochia.)

Operations to effect delivery resulted in this series as follows: In his febrile series there was one woman who had been delivered by forced accouchement who showed gonococci in the lochia; while a second one, upon whom version had been performed, was the case of streptococcic infection. There were 8 others operated upon in the series, and of them, 3 were positive (version, low forceps, and induced labor, of each, 1 case); 3 other cases showed gonococci (2 low forceps and 1 bag induction); of 2 cases, 1 gave on the third day a variety of Bacillus dysenteriæ, while the other gave Micrococcus aureus and Bacillus pseudodiphtheriæ on the seventh day (the first was a midforceps operation, and the latter a case of version).

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The former of these last 2 is considered to have been a contamination at the time of culture.

GENERAL CONCLUSIONS.—In 50 cases studied by Little the uterus was found to be sterile in 92 per cent., 50 per cent., and 44 per cent. on all three days. If gonococci be included, the above averages are 96 per cent., 72 per cent., and 67 per cent. The puerperium is to be noted as normal in 40 and febrile in 10 cases of this series. In the normal cases there was absolute sterility in 92.5 per cent., 62.5 per cent., and 50 per cent., as compared with the febrile series, in which 90 per cent., 40 per cent., and 20 per cent. were found to be sterile. Including the gonococcic cases, these latter figures would be 95 per cent., 85 per cent., and 70 per cent., and 100 per cent., 50 per cent., and 50 per cent. The author considered his results as positive if bacteria were found only in smear preparations or only in cultures, as well as when present in both. It is likely, therefore, that a certain number of positive results were due to contamination and that the uterus is really sterile in a larger number of cases than these percentages would indicate. Streptococcus was present but once—a febrile case, on the third day; absent, however, on the first and seventh days.

Finally, for purposes of completeness, the following schedule of results is worth including, this being taken from Little's article:

						Per cent.
Von Ott	in	9	cases	found lochia sterile in		100
Czerniewski	"	57	"	"	"	98
Doederlein	"	30	"	"	"	90
Doederlein	"	250	"	"	"	83
Vogel	"	15	"	"	"	80
Franque	"	10	"	"	"	80
Kronig	"	63	"	"	"	79
Thomen	"	9	"	"	"	66
Walthard	"	20	"	"	"	65
Stahler	"	55	"	"	"	64
Frans	"	10	"	"	"	0
Burckhardt	"	14	"	"	"	7
Stahler	"	19	"	"	"	16
Wormser	"	100	"	"	"	16
Stols on the fourth day				"	"	19.6
Vogel in		15	"	"	"	34
Schauenstein in		100	"	"	"	36
Stols on the ninth day				"	"	65

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The importance of definite and certain knowledge on this whole question cannot be overestimated, because of its bearing upon the general question of puerperal septic infection and more particularly upon the frequency of auto-infection. If some investigators are correct in their findings of pathogenic organisms in a great proportion of normal cases after delivery, it is very evident that the value of this method as an aid to diagnosis is nothing; and, moreover, that auto-infection is one of the most common causes of puerperal sepsis, instead of being, as is the general opinion at present, one of the most rare happenings. Even if the extreme position of Stolz with regard to the frequency of positive findings be found untenable, proof that the ascent of organisms after the first few days into the cavity of the uterus is a usual or even a moderately frequent event will render useless any further consideration of this matter, since the import of positive findings in any given case will be beyond determination.

The probability that streptococci would be satisfied to act as saprophytes within the uterine cavity after delivery being to us a matter of grave doubt, together with the fact that in normal cases, used for demonstration of the method before the class, the laboratory reported pure streptococcic cultures, turned our attention forcibly to the question of the technique employed in the clinic for the collection of the lochia. It being a matter of common knowledge that the lower vagina in most cases is the seat of bacterial activity, and that in a goodly number the same condition prevails in the cervix and upper portion of the vagina, we felt that the method employed, while as careful as any then known to us, demanded modification if it was to be worth doing at all. At this time the ordinary Doederlein tube was employed, sterilized by boiling, and its introduction was performed with the usual aseptic precautions. Realizing that introduction without contamination from the cervix was certainly impossible, if done several days after delivery, and that it was improbable at any time, it was assumed that a positive culture obtained in a normal case simply meant that there was bacterial growth somewhere below the internal os uteri;

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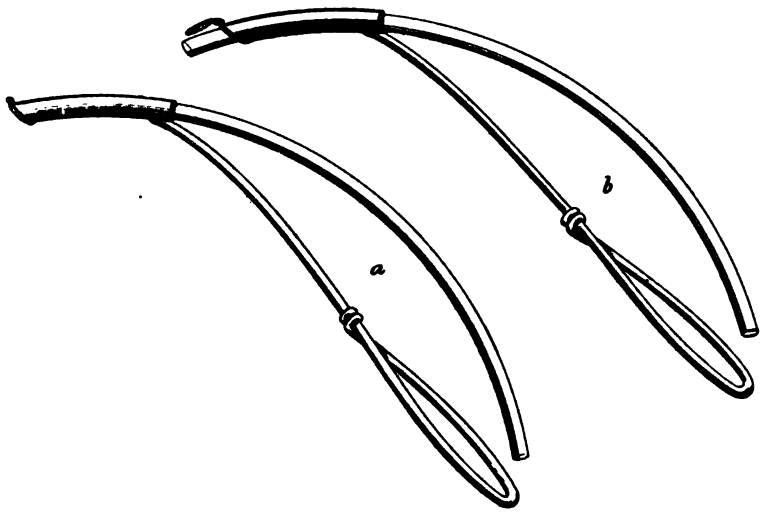
while those in which this method gave negative results simply gave evidence of both a sterile endometrium and an absence of contamination in the vagina and cervix. Our attention, therefore, was directed toward a modification of the technique which would render possible the collection of uterine lochia without the risk of contamination from the lower canal.

After some experimentation it was decided that there had been several weak points in the technique which we had been in the habit of using, and the method upon which the present findings



Curved cervical speculum for taking intra-uterine cultures: *a*, tube with cap closed ready for insertion; *b*, after insertion, showing the open cap and the projected inner tube for collecting the secretions.

are based was evolved. Its most important element (see figure) is the use of a slightly curved cervical speculum, the distal end being closed by a hinged cap. A handle of convenient length is attached to render its introduction more easy. We do not claim originality for this idea, as, although at the time the fact was unknown to us, various forms of protective specula have been employed by others. We believe that our instrument will be found to be the simplest as well as the most trustworthy. To enable us to use the

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speculum, we discarded the typical Doederlein tube and adopted one with a uniform curve and of small caliber, easily obtained from any manufacturer.

The method in detail is as follows: The patient is placed in the lithotomy position and the external genitalia are carefully cleansed with tincture of green soap, water, and mercuric bichloride. A Sims speculum is inserted, the cervix is grasped by a double tenaculum, and its vaginal portion cleansed with cotton and bichloride solution. The metal tube, with cap closed, is then inserted into the uterine cavity. This tube is sterilized by pressure and wrapped in two coverings, in order that the operator may remove it from the inner one without contaminating his hands. The glass tube is placed within the metal one before sterilization and is not removed from the latter until the culture has been completed. The cap is opened by pushing the glass tube into the cavity of the uterus, a piece of sterile rubber tubing is attached to the proximal end of the glass tube, and as much lochia as possible is sucked into the glass tube by means of a hard rubber syringe. The glass tube is then withdrawn until its distal extremity is engaged within the metal tube and then both are simultaneously removed from the patient. The ends of the glass tube are sealed with wax and the tube is sent to the laboratory. The points of paramount importance, in our opinion, are the presence of the cap upon the distal end of the sheath tube and the sterilization of the tubes by pressure instead of by boiling. We also feel that the simultaneous introduction of both tubes is very important and that their simultaneous removal is but little less important.

As important as the collection of the uterine lochia is the technique employed in the laboratory. The danger of outside contamination, especially from the tube itself, must be recognized. For this reason the tube was washed with a bichloride solution, the ends broken off, and the contents placed in a sterile Petri-dish, cultures being made from this upon the following media: (1) Agar-agar stroke plates; (2) glycerin agar stroke plates; (3) serum agar stroke plates; (4) bouillon tubes; (5) litmus milk tubes for anaërobic

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cultures by Wright's method; (6) glucose agar for anaërobic cultures. In addition, coverslip preparations were made, two being stained with Löffler's stain and two with Gram's stain.

At times this routine method was deviated from because of the lack of serum agar. This medium was used for the demonstration of the gonococcus. It has been our experience in studying the gonococcus that in the secretions in which the organism could not be demonstrated microscopically no growth soccurred upon the special medium used for its cultivation, and that often it could be found in the stained specimen without growth upon the artificial medium, owing to the overgrowth of a mixed culture. For this reason we do not believe that our results as to the presence of the gonococcus are vitiated by the occasional omission of this form of medium. In this series the presence of the gonococcus was remarkably infrequent. We believe this to be due to the technique, which eliminated cervical contamination and proved conclusively to our minds an already formed opinion that gonorrheal endometritis is a much rarer condition than is generally thought. We do believe, however, that this organism is present and persists for a long time in the cervix, often acting as the forerunner of septic infection. Two of the cases in this series showed the presence of the gonococcus; the streptococcus was also found. The frequency with which the gonococcus has been found by other observers we believe to be due to cervical contamination.

In determining the qualifications of a normal case, we have differed from some of the other investigators in this field. We have taken no note of lacerations of either the cervix or the perineum, the duration of the labor, the number of vaginal examinations, the parity of the woman, or the presence of evanescent temperature, unless the latter has been shown clearly to be dependent upon pathological conditions in the generative tract. In other words, we have classified a case as normal if there has been no instrumental interference in labor, if the hand has not been introduced into the uterus, if gauze has not been introduced into the uterus or vagina, and if no douches have been given, if there has been no retention of con-

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siderable portions of the placenta or membranes, and if no alteration of the odor or appearance of the lochia has occurred. In short, we use the term normal as descriptive of the most common parturient state. We believe that by so doing we are likely to reach more valuable conclusions than by more closely restricting ourselves in our selection of cases for study, since the important fact for determination is the bacterial content of the uterus in the type of case most usually met with in actual practice.

With regard to the value of the intra-uterine culture as an aid to diagnosis, we feel that it is a method which should not be neglected in the routine study of infected cases, at least in institutions having the advantage of well-equipped laboratories of bacteriology. The technique as regards asepsis is more exacting than that required for successful blood culture, since the vagina and cervix, with their bacterial flora, must be traversed in obtaining the intra-uterine culture. If a suitable technique be followed, we are confident that the results, as a whole, will well repay the time spent in the procedure; but we do not desire to be understood as affirming our belief that the method is, as it was formerly thought, a sovereign means of diagnosticating the presence or absence of infection in all cases. We have, for instance, four records not included in this paper, in which in cases afterward proved to be septic, the intra-uterine findings were negative. That such an occurrence will take place as a matter of course, more or less often in a series of examinations of septic cases, must be evident to anyone who will consider the varied manifestations of septic processes during the puerperium. On the other hand, the relative rarity of this negative finding in the presence of septic infection offers valuable evidence as to the general utility of the method.

Taken in connection with the studies upon the bacteriology of the blood which have been made in our clinic, we feel justified in making the following statements: (1) If both blood and intra-uterine cultures are negative, the temperature is due to some intercurrent condition; (2) if the intra-uterine culture is positive, but the blood culture is negative, we consider the infection to be still local in its manifestations; and (3) if the intra-uterine culture is

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negative but the blood culture is positive—a very unusual condition—we are justified by our general conclusions in considering that the case is one of general infection, the local endometrial condition having been either sterile from the first or having become so gradually after the general infection developed.

It will be noted that we have classified 6 cases as normal which have given cultures in the lochia, and that we have explained the growth in them by errors in technique. We feel that this demands a fuller explanation in order that we may be entirely intelligible. It is necessary that it be clearly understood in the first place that these 6 cases do not include by any means all the cases in which there was a departure from the method described in the first part of this paper. In each instance any such departure was noted at the time the culture was made, and these are the only cases associated with technical error in which a growth was obtained. In the greater number of cases, however, in which accidental departures from the technique occurred, without cultural results, the variation was trivial, being simply noted for reasons of scientific accuracy. In the 6 cases mentioned the variations in the method were of a nature to give expectation of the probability of vaginal or cervical contamination. It is also quite true that the same, or as grave, errors were made in a few cases which did not show positive results in the culture; and this emphasizes our claim that, while a technique as rigid as ours may not aways be necessary to obtain satisfactory results in each individual case, still, if error has been made in technique, it is manifestly unfair to consider positive findings as evidence of intra-uterine infection.

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Normal Cases—Cultures Taken in the First, Second, and Third Periods.

Number.	Case number.	First period: First to third day.			Second period: Fourth to seventh day.			Third period: Eighth to thirteenth day.			Remarks.
		Aërobic.	Anaërobic.	Microscopic.	Aërobic.	Anaërobic.	Microscopic.	Aërobic.	Anaërobic.	Microscopic.	
1	1	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
2	2	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
3	5	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal except for one temperature of 102° on the sixth day (a few hours after culture) falling at once to normal.
4	7	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
5	25	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Student introduced hand into vagina to deliver placenta; otherwise a normal case. On third culture three attempts made before tube passed.
6	56	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
7	57	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
8	58	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
9	61	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
10	62	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Dead foetus delivered at home: otherwise normal.
	34	See error in technique.			Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Labor normal. Error made at first culture—whereby vaginal secretion obtained.

Normal Cases—Cultures Taken in the First and Second Periods.

11	13	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal. Febrile temp. once because of congested breasts.
12	27	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal. One febrile temp.
13	28	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal.
14	41	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal.
15	42	Sterile	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal.
16	43	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal.
17	65	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal.
18	6	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Normal.

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Normal Cases—Cultures Taken in the First and Third Periods.

19	9	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
20	14	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
21	15	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
22	22	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
23	59	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
24	69	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Steri e.	Negative.	Normal.
25	70	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
	51	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.
	20	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Normal.

Normal Cases—Cultures Taken in the First Period Only.

26	19	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal.
27	60	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal. Temperature febrile second and third day.
28	29	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal.
29	31	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal.
30	44	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal.
31	38	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Abortion at 6 months. Breech delivered—Wiegand.
32	39	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal.
33	64	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Prolapse of cord (no version). Otherwise normal.
34	4	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Normal.

Normal Cases—Cultures Taken in the Second and Third Periods.

35	10	....	....	....	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Tuberculosis. Irregular febrile temperature.
36	16	....	....	....	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Normal.
	55	....	....	....	Sterile.	Sterile.	Negative.	See error in technique.			Normal.

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Normal Cases—Cultures Taken in the Second Period Only.

Number.	Case number.	First period: First to third day.			Second period: Fourth to seventh day.			Third period: Eighth to thirteenth day.			Remarks.
		Aërobic.	Anaërobic.	Microscopic.	Aërobic.	Anaërobic.	Microscopic.	Aërobic.	Anaërobic.	Microscopic.	
37	47	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Normal.
38	48	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Normal.
39	49	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Normal.
40	53	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Slight phlegmasia.
41	37	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Normal. Temperature once 101.2°
42	52	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Normal.
43	66	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Normal.

Normal Cases—Errors in Technique with Positive Findings.

44	34	Mic. pyog. aur., Bac. xeroides, Mic. magnus.	No obligatory anaërobe.	Gram positive micrococci and bacilli.	See normal cases: Cultures taken in first, second, and third periods.						The first culture was taken as a demonstration. The tube became blocked and was accidentally withdrawn and vaginal secretion inspirated.
45	20	See normal cases: Cultures taken in first and third periods.			....	....	....	Mic. pyog. alb.	No obligatory anaërobe.	Gram positive micrococci.	On last culture tube was introduced without speculum. This growth considered vaginal or cervical contamination.
46	51	See normal cases: Cultures taken in first and third periods.			....	....	....	Mic. pyog. aur.	No obligatory anaërobe.	Gram positive micrococci.	The last culture was taken by resident physician and mistake in technique was made.
47	24	Streptococcus, Bac. coli com.	No obligatory anaërobe.	Gram positive micrococci. Gram negative bacilli.	....	....	....	....	....	....	Attempt to pass tube failing, it was withdrawn and passed by the aid of vaginal touch. Case normal clinically.
48	54	....	....	....	Mic. pyog. alb.	No obligatory anaërobe.	Gram positive micrococci.	....	....	....	Tube boiled and passed without speculum.
49	55	....	....	....	....	....	....	Mic. pyog. alb. bac.	No obligatory anaërobe.	Gram positive micrococci.	Tube passed without speculum; cervix not cleansed; cap opened in vagina.

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Abnormal Cases Obstetrically—Forceps.

50	45	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Low Sawyer forceps.
51	18	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	....	....	....	Simpson forceps. Slight fever on third day.
52	63	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Simpson forceps. Bad cervical tear. Much clotted blood in vagina at first examination. Vaginal discharge foul. Intra-uterine douche several times before last culture. Leukocytes, 14,000. Blood culture, negative.
53	40	Sterile.	Sterile.	Negative.	....	....	....	....	....	....	Low forceps. Temperature never febrile.
54	50	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Simpson forceps. Temperature never febrile.

Abnormal Cases Obstetrically—Douches; Manual Removal of the Placenta; Version; Intra-uterine Pack

55	23	Sterile.	Sterile.	Negative.	....	....	....	Sterile.	Sterile.	Negative.	Temperature never febrile. Intra-uterine douche before cultures.
56	17	Mic. pyog. alb.	No obligatory anaërobe.	Gram positive micrococci.	....	....	....	Sterile.	Sterile.	Negative.	Adherent placenta. Manual removal. Normal temperature.
57	35	....	....	....	Sterile.	Sterile.	Negative.	Sterile.	Sterile.	Negative.	Placenta previa with version. Gauze pack. Irregular fever. Lochia foul. Complete tear. Child macerated. Blood culture negative.
58	46	....	....	....	Sterile.	Sterile.	Negative.	....	....	....	Intra-uterine pack.

Abnormal Cases Obstetrically—Craniotomy.

59	30	Mic. pyog. alb., Bact. coli com.	No obligatory anaërobe.	Gram positive micrococci. Gram negative bacilli.	Mic. pyog. alb., Bact. coli com.	No obligatory anaërobe.	Gram positive micrococci. Gram negative bacilli.	....	....	....	Never febrile. Craniotomy. Fetid discharge. Probable muscular rupture of uterine wall.
60	11	....	....	....	Mic. magnus., Mic. pyog. aur.	No obligatory anaërobe.	Gram positive micrococci.	....	....	....	Craniotomy for hydrocephalus Normal temperature.

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Intercurrent Disease—Typhoid Fever

Number	Case number.	First period: First to third day.			Second period: Fourth to seventh day.			Third period: Eighth to thirteenth day.			Remarks.
		Aërobic.	Anaërobic.	Microscopic.	Aërobic.	Anaërobic.	Microscopic.	Aërobic.	Anaërobic.	Microscopic.	
61	33	....	....	....	. ..	....	....	Sterile.	Sterile.	Negative.	Douche before admission. Blood culture. Bac. typhosus.

Gonorrhœal Cases.

62	12	Mic. pyog. aur.	No obligatory anaërobe.	Gram positive micrococci. Gonococci.	Mic. pyog. aur.	No obligatory anaërobe.	Gram positive micrococci. Gonococci.	....	....	....	Child developed ophthalmia neonatorum. Never febrile.
63	26	Sterile.	Sterile.	Gonococci.	....	....	....	Sterile.	Sterile.	Gonococci.	Douche before second culture. Febrile after first culture. Diagnosis: Gonorrheal rheumatism and breast abscess.
64	67	Streptococcus.	No obligatory anaërobe.	Gram positive micrococci. Gonococci.	Sterile.	Sterile.	Negative.	....	....	....	Normal temperature.

Septic Cases.

65	3	Sterile.	Sterile.	Negative.	Streptococci	No obligatory anaërobe.	Gram positive micrococci.	....	....	....	Blood culture showed streptococcus pyogenes. Febrile third to fourteenth day. Breast congested. Many intra-uterine douches.
66	68	Streptococcus.	No obligatory anaërobe.	Gram positive micrococci. Gonococci.	Streptococci	No obligatory anaërobe.	Gram positive micrococci. Gonococci.	....	....	....	Febrile 3 days. Manual extraction placenta. Purulent cervical discharge. Diagnosis puerperal endometritis.
67	21	....	....	....	....	....	....	Streptococcus.	....	Gram positive micrococci.	Blood culture: Steptococcus pyogenes.

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To consider these 6 cases from the standpoint of actual error occurring in each case:

CASE XXXIV.—The first culture showed Micrococcus aureus and magnus and Bacillus xeroides. The case was used for demonstration, the tube accidentally slipping, whereby vaginal secretion was inspirated.

CASE XX.—First culture sterile; second culture gave Micrococcus albus. Here the variation consisted in the omission of the Sims speculum in the introduction of the tube, it being guided into the cervix by the finger in the vagina.

CASE LI.—In this case the first culture was sterile, while the second, taken by an assistant, showed a growth, Micrococcus aureus. In both instances the variation in technique was identical, the vaginal speculum being omitted. From our knowledge of the ease of contamination, we consider that this second culture was contaminated by lack of care upon the part of a man inexperienced in the method. We admit that this contention is not proved.

CASE XXIV.—But one culture was made, and this gave the streptococcus and the colon bacillus. In this instance the Sims speculum prevented sufficient depression of the tube to enable it to pass, so the former was discarded and the tube was passed by guidance of the finger. During the short time elapsing between the introductions, the tubes were both placed in the instrument tray, which was an additional source of possible infection.

CASE LIV.—Tube was introduced by guidance of a finger in the vagina. In addition, the tube was sterilized by boiling instead of by pressure.

CASE LV.—Tube was introduced, as in the last case, and, in addition, the cap of the metal tube opened while it was in the vagina during the introduction.

It is to be noticed that the omission of the Sims speculum was noted in each instance in which in a normal case a growth was recorded. In two instances there were other factors present which would have been sufficient to cause contamination—in one case the protective cup was open during introduction, while in the other both tubes lay for several minutes in an instrument tray the sterility of which during that period must be considered doubtful.

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Because of difficulties inherent to the work it was not possible, and, indeed, it did not seem desirable, to culture each case upon the same day of the puerperium, so we have divided the cultures into three periods. We have further divided the whole list of cases under various headings as follows:

A. Clinically normal cases:

1. Cultures during first period (first to third day), 34 in number; all sterile.
2. Cultures during second period (fourth to seventh day), 29 in number; all sterile.
3. Cultures during the third period (eighth to thirteenth day), 18 in number; all sterile.

B. Clinically normal cases with errors in technique and positive results:

1. Cultures during first period, 2 in number.
2. Cultures during second period, 1 in number.
3. Cultures during third period, 3 in number.

(All of these cases, with one exception, in which but one culture was taken, showed negative results from other cultures made without error.)

C. Clinically abnormal cases:

(*a*) Forceps (in excavation or at outlet), 5 cases.

1. Cultures during first period, 4 in number; all sterile.
2. Cultures during second period, 3 in number; all sterile.
3. Cultures during third period, 1 in number; all sterile.

(*b*) Douches (intra-uterine), 1 case:

1. Cultures during first period, 1 in number; sterile.
2. Cultures during third period, 1 in number; sterile.

(*c*) Manual removal of placenta, 1 case.

1. Cultures during first period, 1 in number; growth.
2. Cultures during third period, 1 in number; sterile.

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(*d*) Craniotomy, 2 cases:

1. Cultures during first period, 1 in number; growth.
2. Cultures during second period, 2 in number; growth.

(*e*) Version for placenta prævia (macerated fœtus):

1. Cultures during second period, 1 in number; sterile.
2. Cultures during third period, 1 in number; sterile.

(*f*) Uterine packing:

1. Cultures during second period, 1 in number; sterile.

D. Intercurrent disease:

Typhoid fever (blood positive).

1. Cultures in third period, 1 in number; sterile.

E. Gonorrhœa:

1. Cultures during first period, 3 in number; all positive.
2. Cultures during second period, 2 in number; 1 positive.
3. Cultures during third period, 1 in number; positive.

One case, positive in the first and third periods, was unassociated with other organisms. In this case blood examinations were negative, but a mammary abscess and gonorrhœal rheumatism were complications.

The second case, positive in the first and second periods, was associated with Micrococcus aureus in both cultures; while the third case, positive at the first culture and associated with streptococci, was negative in the second period and also sterile at this time.

F. Septic cases (3):

1. Cultures during first period, 2 in number; positive.
2. Cultures during second period, 2 in number; positive.
3. Cultures during third period, 1 in number; positive.

(Cultures not taken of third case during first or second period.) Blood cultures were positive in 2 of the above cases, and were not taken in the third case.

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GENERAL CONCLUSIONS.

1. The uterine lochia is sterile in normal cases throughout the puerperium.

2. Streptococci are never present within the cavum uteri without causing symptoms.

3. In a few instances non-pathogenic germs may be found in cultures in afebrile cases, but there is every reason to believe that their presence is really the result either of contamination during the extraction of the lochia or of their introduction during obstetric manipulation.

4. Ascendance of the gonococcus is an event of comparative rarity, though the frequency of this organism would of itself give reason to expect otherwise.

5. Infection of the endometrium is an ever present danger in culturing within a few days of delivery.

6. A study of the bacterial content of the puerperal uterus is of great importance as a subsidiary means of diagnosticating septic infection following delivery.

7. As nearly as may be, a technique should be adopted which will prevent contamination during the removal of the lochia in order to avoid a vitiated result.

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CARDIAC ARRHYTHMIA.[1]

FROM A PRACTICAL STANDPOINT, IN THE LIGHT OF RECENT INVESTIGATIONS.

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Before entering upon a description of the various forms of cardiac arrhythmia it may be profitable to review hastily a few of the basic tenets of the myogenic theory of cardiac activity, since the modern studies of arrhythmia are based upon this conception. The myogenic doctrine maintains that the rhythmic contraction of the heart is due primarily to inherent properties of the cardiac muscle, and relegates to a position of secondary importance the nervous and chemical stimuli which doubtless play a part in the regulatory mechanism.

The heart muscle possesses five distinct properties, which may vary independently of each other: automatic stimulus production, conductivity, irritability, contractility, and tonicity. By *chronotropic influences* are meant those which influence the production of motor stimuli; when positive they accelerate stimulus production, shortening the time required, and thus increasing the rate; when negative the opposite effect is produced. When primary, stimulus production is accelerated directly; when secondary, the results are brought about through changes in irritability or conductivity. *Dromotropic influences* affect conductivity; *bathmotropic*, irritability; and *inotropic*, contractility. For example, stimulation of the pneumogastric nerve produces arrest of the heart. This may

[1] Read April 1, 1908.

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result from cessation of stimulus production (primary negative chronotropic influences), or through depression of excitability (primary bathmotropic influence), or to the fact that the stimulus though normally generated and received is not conducted (negative dromotropic influence).

The heart muscle has the ability to generate rhythmic stimuli, the nature of which is at present unknown, though a chemical character has been suggested, and not without strong reason, for recent experiments have demonstrated the necessity of certain inorganic salts in the maintaining of heart rhythm. Normally these stimuli arise in the venous ostia of the right heart, and pass downward through the bundle of His to the ventricles, not, however, as the result of nervous conduction, but by reason of muscular continuity, from cell to cell (Gaskell, Engelmann). Stimuli, however, may be generated at other points, but under normal conditions they are not of sufficient intensity to interfere with the rhythm, as may be the case pathologically, when the route of conduction may even be reversed. The left auricle, however, seems to have no power of automatic rhythmicity. The wave of contraction passes through different parts of the heart at a varying rate, a condition which favors a more complete ventricular systole (Engelmann). Stimulus production and conduction, as well as contractility, are much influenced by the phase of the cardiac cycle, the function of each being depressed during systole, and gradually restored during diastole. All these functions may vary in activity, independently of each other; they are also much influenced by the pabulum supplied by the arteries and lymphatics, by the removal of waste products through the veins, and by nerve impulses. Stimulation of the vagus depresses stimulus production, conductivity, and contractility. Stimulation of the accelerator nerve has the opposite effect. The vagus acts very slightly if at all on the ventricles, except indirectly through the auricles (Erlanger). In complete heart block the administration of atropine, though increasing the auricular rate, will have no effect on the ventricular rhythm (Gibson and Ritchie). Heart block itself may result from vagus action; this as has been shown by Mackenzie may occur during deglutition. Conductivity is depressed

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by asphyxia; and when this function is below par the administration of digitalis still further increases this depression, a practical fact of great clinical importance, to which I shall again allude.

From the foregoing brief review it is easy to see that in order to explain satisfactorily and study the different forms of arrhythmia we must first reduce them to their simplest terms, so to speak—that is, classify and group them according to whichever one of the five fundamental properties of the heart muscle is chiefly or entirely concerned in their production.

Experimental physiology has made such rapid strides of late years that it is well-nigh impossible for the general practitioner of medicine to keep pace with its latest developments, but our knowledge of the physiology of the circulation has undergone such extensive and important basic changes of late (through the labors of Gaskell, His, Erlanger, Engelmann, Hering, Mackenzie, Cushny, Wenkebach, and others), changes which are daily receiving corroboration and indorsement, that the physician can no longer afford to be uninformed of at least their fundamental principles. The myogenic theory of cardiac activity, while not as yet accepted by all physiologists, is daily gaining adherents. Although it doubtless still requires an enormous amount of investigation and revision, nevertheless, it elucidates satisfactorily and with such minute exactness so many hitherto unexplainable conditions, such as Adams-Stokes disease, extrasystolic arrhythmia, etc., that its acceptance as a working basis is practically necessitated. It was in a very similar manner that Wasserman's theory regarding the blood complement, etc., led to a much more accurate and satisfactory knowledge of the question of immunity than had previously obtained.

The first type of pulse irregularity to which I wish to call attention is:

1. Juvenile Arrhythmia. It is, of course, well known that respiratory activity influences the pulse. In normal individuals inspiration increases and expiration decreases the rate. Some people show this phenomenon much more than others. It is especially common in youth, and has hence been called by Mackenzie the youthful type of arrhythmia. The type of irregularity affects

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chiefly the diastolic period, and for this reason it is noted especially when the pulse is slowing down, after infancy, and after a febrile attack. Mackenzie found that all children at some time exhibited this symptom. It occurs most frequently between the ages of eight and fifteen years, becoming less common toward puberty. It is pathological only when induced to a marked degree by quiet breathing, or when it is not controlled by apnœa. It indicates nerve irritability and is proof of vagus activity. It occurs in fevers, especially during convalescence, in neurasthenia, and in cerebral lesions causing irritation of the vagus. Tracings taken from the precordial region show that it consists of a true intermission, and is not simply a missed beat at the wrist resulting from extrasystole. Needless to say, it has an altogether entirely different genesis than the pulsus paradoxicus, which is entirely due to the mechanical relations of the pericardium and mediastinum. Peters has shown that the vast majority of arrhythmias occurring during the late stages of diphtheria and scarlet fever are of the respiratory type. These two infections are so frequently the cause of serious myocardial lesions that pulse irregularities following them are naturally regarded with alarm. If, however, in such a case we are able to demonstrate a simple respiratory arrhythmia, our prognosis will of necessity be much affected by the finding.

The second group of arrhythmias which I shall consider is the one most frequently encountered, namely:

2. Extrasystole. As a rule, an intermittent pulse is due to extrasystoles, resulting from abnormal irritation at some point of the musculature, generally the ventricle, although it may result from heart block or diminished excitability. These extrasystoles can often be heard on auscultation and are registered on the cardiogram, although the pulse wave may not reach the periphery. The later the extrasystole occurs in diastole the louder will be the sound heard, and the larger the pulse wave, so that if it occurs shortly before the normal time we may get the simulation of a normal, somewhat anticipated, systole. The futile contractions (frustrane Kontraktionen) of Hochhaus and Quinke are examples of extrasystoles occurring early in the diastolic period. Extrasystoles may

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also occur without causing dropped beats or compensatory causes, in cases of bradycardia in which the refractory phase is short in comparison with the length of the pulse rhythm. Experimentally an extrasystole may be produced by increasing the *vis a fronte* in the circulation; it is also seen in digitalis poisoning, and after the administration of calcium. It may also be produced by mechanical and electrical means.

Extrasystoles are explained by the fact that as the refractory phase of the heart rhythm is coming to a close the muscle becomes more and more susceptible to stimuli. An abnormal stimulus sets off the contraction before its time, just as hot carbon in the cylinder of a gasoline engine explodes the charge before sufficient compression has occurred. As the result we see a weak, early contraction which, owing to an insufficient volume of blood and inadequate contractile force—resulting from shortened diastole—barely, if at all, opens the aortic valves. This extrasystole, however, throws the heart into a second refractory period at a time when the normal physiological stimulus should occur; in this manner a beat is dropped and a compensatory pause is brought about. By reason of the latter the number of heart beats per minute may remain unchanged despite the occurrence of extrasystoles, and the length of time occupied by the normal contraction and the extrasystole plus their respective pauses may occupy an equal interval of time as would two normal contractions. This condition of affairs is characteristic of extrasystoles arising in the ventricle. When the abnormal stimulus arises at the mouths of the veins in the right heart, the compensatory pause is absent; if in the auricle itself, it may or may not be present; if in the ventricle, it is present and is not infrequently prolonged.[1] But if this prolongation is great we are strongly led to suspect that the trouble is due to depression of conductivity and not to the fact that the stimulus arrived in the refractory period.

A series of extrasystoles following each other gives rise to the pulsus bigeminus, trigeminus, quadrigeminus, etc., according to their frequency, while a continuous series may produce a clinical paroxysmal tachycardia, although Hoffmann has shown that in tachycardia

[1] Hering, Pflüger's Arch., lxxxii; Präger med. Wooh., xxvi, Nrs. 1 und 2.

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due to this cause a series of compensatory pauses may be distinguished.

Extrasystole is often recognized subjectively by the patient as a dropped or a doubled beat, or a powerful thump which follows the compensatory pause. It is not however, always an easy matter to determine whether an arrhythmia is or is not due to extrasystole. Thus, when the intermissions occur in different kinds of groups, with varying degrees of peripheral pulsation; when we find abnormally long compensatory pauses, and small pulses following them, we may be unable to make out the regularity of the rhythm both in the ventricle and in the artery, despite the fact that the stimulus production may be going on normally at the venous ostiæ. Furthermore, extrasystoles often complicate other forms of arrhythmia, making interpretation difficult. We have the three definite varieties of extrasystoles: (1) Auricular; (2) ventricular; and (3) auriculoventricular; the latter two being the more common. What is known as the interpolated ventricular extrasystole is one which has but little influence on the rhythm, although it may produce a retrograde auricular systole.[1]

Extrasystole is seen in many conditions, which may be grouped as follows: (1) Physiological or individual; (2) after severe exertion; (3) in women at puberty, in pregnancy, and at the climacteric; (4) toxic causes: alcohol, ether, tobacco, infectious fevers, especially during convalescence, and in intestinal disorders; (5) nervous disturbances: neurasthenia, hysteria, neuroses; (6) arteriosclerosis; and (7) organic heart disease.

Clinically, extrasystoles are generally manifested as two or more approximated beats, followed by an intermission. If the extrasystole occurs early in the diastolic period, as is generally the case, we, as a rule, hear on ausculatation a muffled third sound during diastole. The heart is at this time insufficiently filled with blood, the pressure in the aorta is still high, and the ventricular contractile power weak, owing to insufficient rest, as a result of which the aortic valves are not opened by the extrasystole. If the extrasystole occurs

[1] Hering, XXIII Kongr. f. inn. Med., München, 1906.

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later in the diastolic period, a second sound may be produced. The extrasystole is generally followed by an unduly prolonged silence, which is, in turn, brought to a close by a strong, loud, muscular sound of the next regular systole. If the extrasystole has resulted in opening the aortic valves, a small, early pulse wave is seen on the sphygmogram—pulsus bigeminus. Rarely the tracing may simulate the normal dicrotic wave.

Extrasystoles may be induced by a number of causes. It has been shown, both experimentally and pathologically, that increased aortic blood pressure is a prolific source of this symptom. Extrasystoles are also brought about through pathological changes in the myocardium, which appear to initiate a stimulus to which the neighboring normal muscular fibers respond. Increased excitability—positive bathmotropic influences—toxic or thermic in origin, are also often productive factors. And finally, we have to include nervous influence. The latter influence is an indirect one acting through vasoconstriction, which is a well-known excitant. Extrasystoles often occur as an early sign of functional derangement in patients with obesity or arteriosclerosis, and although they may exist for years without any more serious symptoms, their presence calls for careful investigation of the case in which they appear. According to Hewlett, extrasystoles usually do not develop during the acute myocardial lesions of infectious diseases, but when they do appear under such conditions they are of a relatively worse significance.

The occasional occurrence of extrasystole in young, otherwise healthy individuals is of little importance. The symptom, however, always points to cardiac irritability, and while this may be normal for some individuals, its presence should always make us look carefully for intracardial and extracardial abnormalities. Anyone who has auscultated the heart in horses or dogs is aware how frequently extrasystoles occur, and how easily they are produced in these animals. Similarly there are certain individuals, notably those with highly strung nervous systems, who exhibit the symptom more or less constantly. Beyond indicating cardiac irritability, extrasystoles give us no positive clew; they are not pathognomonic of any particular disease.

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Wenkebach divides individuals exhibiting extrasystoles into four groups:

1. People who live to old age without demonstrable heart lesion, who have experienced this symptom off and on for a variable period of years, without any deleterious consequences. Such cases probably result from a congenital or acquired hypersusceptibility to stimuli.

2. The extracardial group, to which class belong the toxic and reflex cases. Extrasystoles are very commonly seen during the convalescence from infectious fevers—typhoid fever, pneumonia, diphtheria, etc. They also occur as a result of the use of tobacco, alcohol, tea, and coffee; as well as in cases of gastro-intestinal disturbances, intestinal parasites, etc.

3. To this class belong those who are the subjects of circulatory disease, regardless as to whether the heart be seriously affected or not. For instance, cases with arterial hypertension frequently exhibit extrasystoles. The same may be said of arteriosclerotics. In these cases the symptom is often intermittent and readily produced by physical effort or mental excitement. Some of these cases can, of course, be explained on the basis of hypertension, but Wenkebach favors the view that the cardiac muscle becomes more irritable as age advances, for the following reasons: (*a*) Other signs of irritability, such as delirium cordis, are frequent in elderly people. (*b*) In both young and old subjects extrasystoles are of common occurrence when arterial tension is low. (*c*) Diseases in which hypertension is most frequent, such as chronic nephritis, are not those in which extrasystoles are most common.

4. Cardiopaths. In this class extrasystoles are of frequent occurrence. We can, however, draw no definite deductions by the mere fact of their presence as to the stage, character, or extent of the heart lesion, Productive factors are present in great numbers in such cases, that is, dilatation, inflammation, local cardiac anemia, overexertion, etc. The worst cases of heart disease often terminate fatally without having at any time exhibited extrasystoles. Wenkebach concludes: After all that has been said, clinicians ought not to attach too much significance to extrasystoles in themselves, and yet they ought to consider it worth while to examine every case and

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determine whether a cardiac lesion is present or not, whether there are any conditions (and they must often be looked for outside of the heart) present, which could account, directly or indirectly, for the presence of the extrasystoles.

Mackenzie, basing his views on the postmortem histological findings of Keith, states that in the majority of cases of extrasystole in the aged there are to be found minor pathological changes in the auriculoventricular bundle which can account for the de-

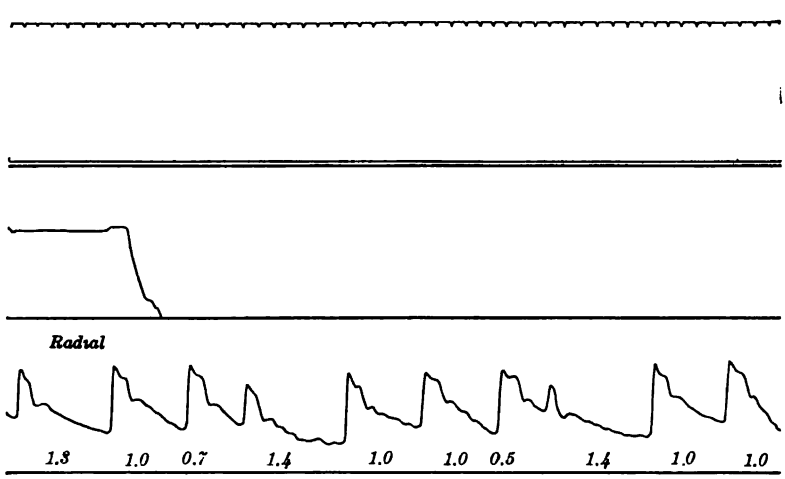

FIG. 1.—Extrasystoles of ventricular origin, followed by compensatory pauses. The normal systole plus the extrasystole occupy the same or a slightly longer interval of time than two normal beats.

pressed conductivity and hyperexcitability, although he admits that there were also present abnormalities of the coronary arteries and of the ventricular musculature.

The accompanying illustration (Fig. 1) shows extrasystoles of ventricular origin occurring in a patient with arteriosclerosis. It was present only temporarily after a change of posture. It appeared in the erect and in the recumbent position for a few minutes after the posture was assumed, and then gradually ceased. The systolic blood pressure was 135 mm., the diastolic 95 mm.[1]

[1] The time recorded on all of the following tracings is in units of one-fifth of a second

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Fig. 2 exemplifies the occurrence of extrasystoles of auricular origin. They are preceded by auricular (*a*) waves in the jugular tracing, and are not followed by compensatory pauses, as are ventricular extrasystoles. This tracing was taken from a man aged sixty-six years, with a high grade of arterial thickening, and a systolic mitral murmur which completely replaced the first sound of the heart. The heart dulness was greatly enlarged to both right and left. The systolic pressure was 160 mm., the diastolic 110 mm. He complained of vertigo after any sudden change of posture, precordial oppression with dyspnœa on exertion, and improved under the

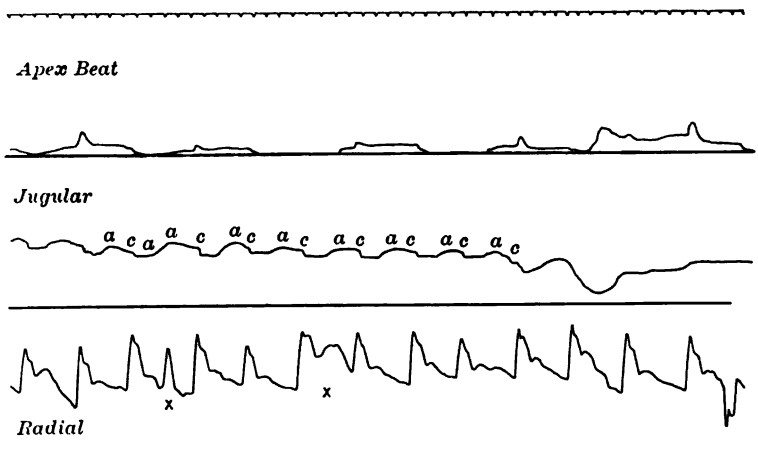

FIG. 2.—Extrasystoles of auricular origin. They are preceded by auricular waves in the jugular tracing *a*, and are not followed by compensatory pauses.

administration of digitalis. There is marked delay in conductivity —the *a* to *c* interval being 0.4 instead of 0.2 of a second, indicating myocardial involvement.

Fig. 3 illustrates extrasystoles of auriculoventricular origin; in other words, the abnormal stimulus arises in the bundle of His. The large waves marked *a* and *c* are due to the simultaneous contraction of the auricles and ventricles, both being premature. These waves are not followed by a *c* wave, and the diastole following is of the same length as the normal auricular rest period. This is the form of extrasystole which has been designated by some observers as

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the retrograde extrasystole, it being assumed that the abnormal stimulus arises in the ventricle and is transmitted backward to the

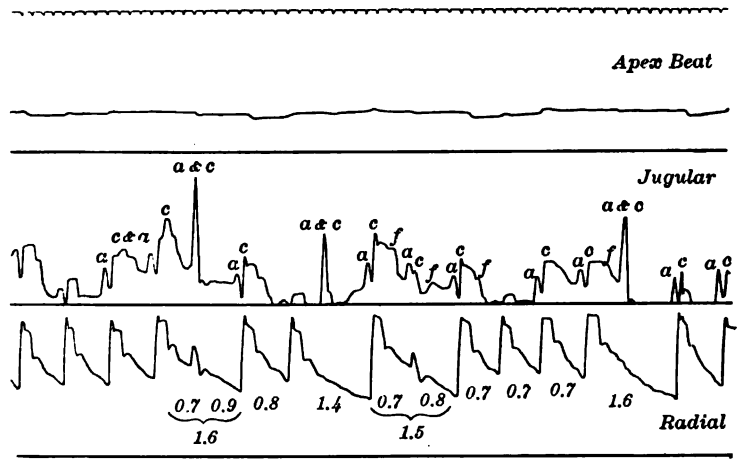

FIG. 3.—Extrasystoles of auriculoventricular origin (arising in the bundle of His). The tall waves *a* & *c* are due to the simultaneous contraction of the auricles and ventricles. They are not followed by *c* waves or by compensatory pauses.

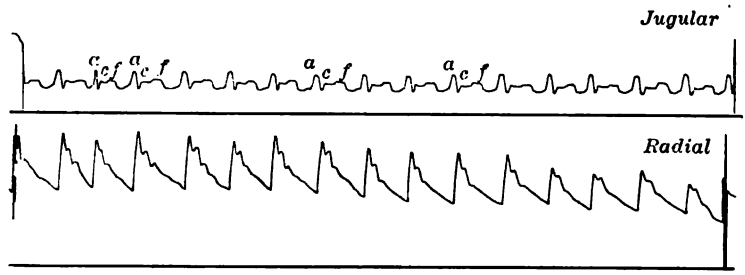

FIG. 4.—The same case as Fig. 2. The extrasystoles have disappeared. The jugular tracing shows the three normal waves.

auricle. This explanation is hardly satisfactory, however, for, as Mackenzie has pointed out, if such were the case there should be

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a delay of one-tenth of a second in the appearance of the auricular wave—the time required for the stimulus to travel across the auriculo-ventricular bundle—whereas, in reality, the contraction of the auricle and ventricle are exactly synchronous.

This tracing was taken from a physician aged sixty-six years, who complained of precordial oppression, and a thumping in his chest which occurred with each intermittence of the pulse. Physical examination disclosed a double aortic lesion. The radial pulse on the tracing shows the characteristically rapid collapse which follows the straight, high, systolic limb.

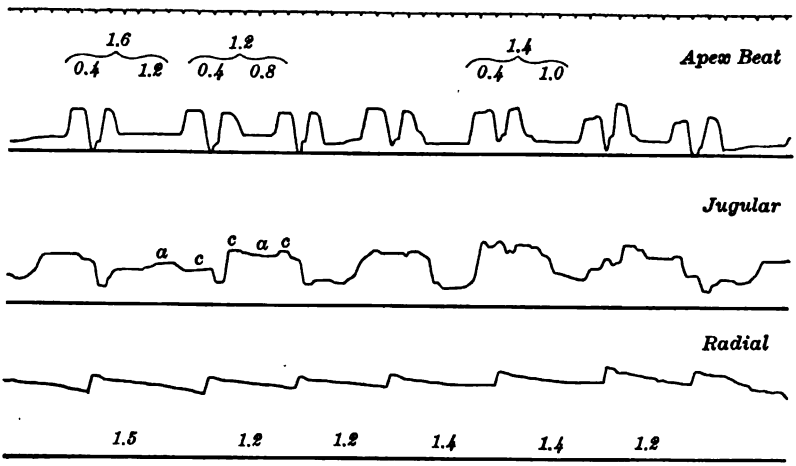

FIG. 5.—Extrasystoles of ventricular origin, which are manifest at the apex beat but do not reach the periphery. Only every other beat causes a radial pulse, thus producing a false bradycardia which without the tracing might have been construed as due to heart-block.

Fig. 4 was taken from the same patient one year later. The arrhythmia had entirely disappeared. The pulse rate was 68, the systolic pressure 190 mm., the diastolic 110 mm., and the patient was comfortable and attending to his work.

Fig. 5 illustrates extrasystoles of ventricular origin, appearing early in diastole, followed by compensatory pauses, and failing to appear in the radial pulse, except occasionally, this being due to the fact that the ventricle has been insufficiently rested and inade-

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quately filled with blood, as the result of which the aortic valves were barely if at all opened. This tracing was taken from a patient with failing compensation—a double mitral lesion, with marked cardiac dilatation; death occurred about two weeks later. On auscultation two fairly loud, close-coupled sounds were heard over the apical region, followed by only one second sound at the base (occasionally two sounds were heard at the aortic area, the second being muffled and indistinct). The pulse at the wrist was for the most part quite regular, but only one beat was felt, while two sounds were heard at the apex area. If the ventricle had been stronger we should have had a typical pulsus begeminus in the radial.

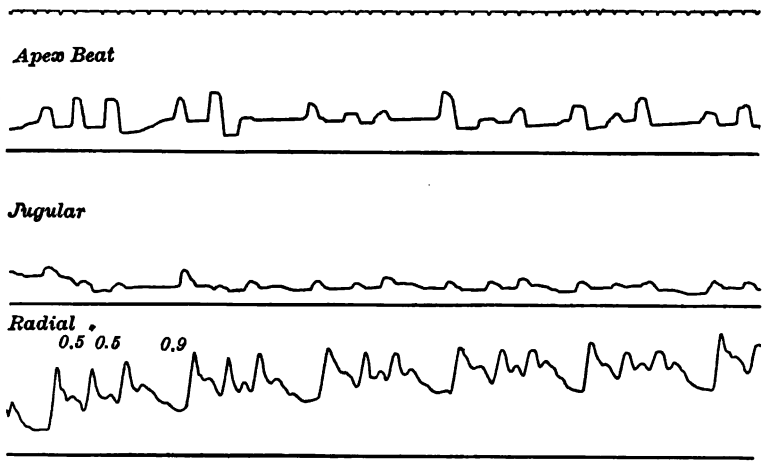

FIG. 6.—Pulsus trigeminus, resulting from extrasystoles occurring in groups of two The jugular pulse is of the positive type—the waves are synchronous with the ventricular systole; taken from a case of mitral and tricuspid insufficiency.

Fig. 6 shows a series of extrasystoles occurring in groups of two, producing a pulsus trigeminus, which occurred in a case of acute endocarditis, which terminated fatally about ten days later. At autopsy in addition to cardiac hypertrophy and dilatation ulceration of the aortic valves was found.

The following tracings illustrate most graphically the way in which digitalis improves the character of the pulse by suppressing the extrasystoles, producing a longer diastole, a more complete

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period of rest, and resulting in a greatly improved state of the circulation. Fig. 7 was taken from a case of mitral stenosis and insufficiency, with cardiac dilatation and tricuspid insufficiency. There was general anasarca, cyanosis, orthopnœa with blood-tinged expectoration, and a pulsating liver. The tracing shows the marked arrhythmia complicated by extrasystoles, and a positive venous pulse. The patient was bled ten ounces and given three drams of the infusion of digitalis thrice daily. A few days later the general condition was greatly improved. At this time the second tracing was taken (Fig. 8), which shows a much slower, better filled and sustained

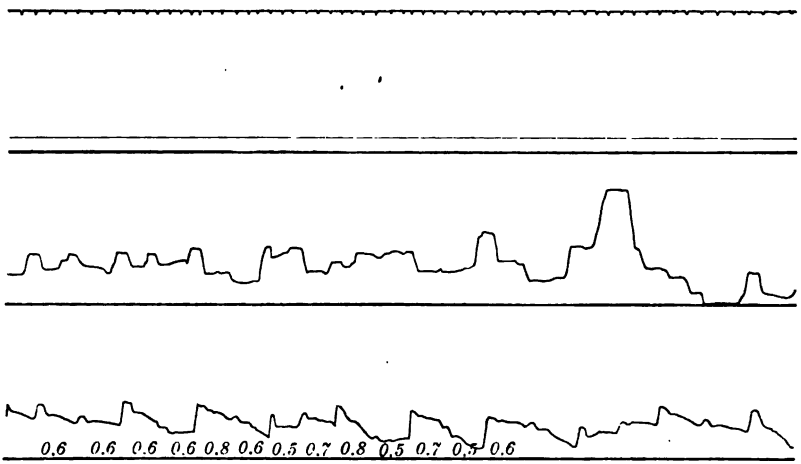

FIG. 7.—Double mitral disease with tricuspid insufficiency, showing extrasystoles and a positive venous pulse.

radial pulse, and many fewer extrasystoles. It will be seen that the jugular pulse is still of the positive type resulting from the tricuspid leakage.

The next type of pulse irregularity to which I would call attention is a variety which is met with quite frequently in the advanced stages of heart disease, generally with failing compensation:

3. PERPETUAL ARRHYTHMIA. Perpetual arrhythmia is one in which no regular rhythm whatever can be made out for long periods of time—weeks or months. The subject of its etiology requires further elucidation. When present in its typical form three charac-

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teristics are noted: (1) The jugular tracing shows no auricular wave; (2) it is always associated with a positive venous pulse; (3) absolutely no underlying rhythm can be detected. As to the second qualification—the positive venous pulse—the majority of authorities hold that this is due to tricuspid insufficiency or to paralysis of the auricle. As to the etiology of this variety of irregularity, Wenkebach believes that it is due to a block between the sinus and the auricle, the result either of muscular weakness or depression of conductivity, and not to auriculoventricular extrasystole or to patho-

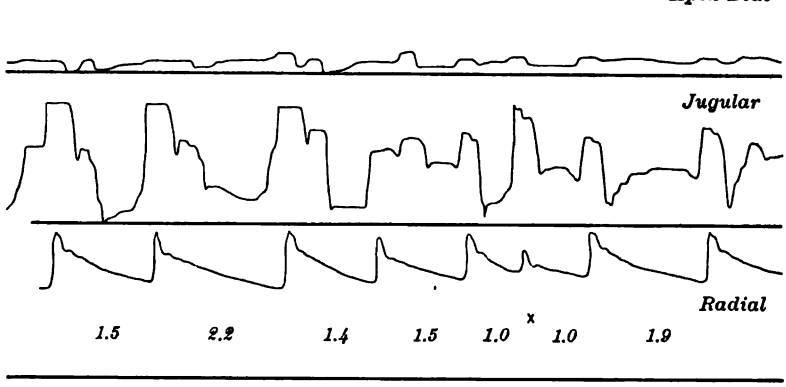

FIG. 8.—Tracing from the same patient as Fig. 7, after venesection and digitalis. Most of the extrasystoles have dropped out. There is a marked improvement in the character of the radial pulse, and there was a corresponding amelioration in the patient's condition. *x*, extrasystole.

logical changes in the auricular muscle. Erlanger has recently performed some experiments which prove the possibility of an experimental sino-auricular block.

When a tracing from a case of perpetual arrhythmia is examined we are struck by the fact that no two pulse waves are of equal length. Clinically the condition is often spoken of as delirium cordis, but as Hewlett has recently pointed out, this character of pulse is sometimes met with even if the pulse is slow. We have already mentioned the fact that a permanently irregular pulse with-

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out tachycardia or bradycardia, unaffected by respiration, digitalis, or atropine, is always associated with a ventricular venous pulse. Since auricular paralysis or tricuspid insufficiency is practically only encountered in cases with broken compensation, we find the permanently irregular pulse only in very serious cases. It is apparently always intracardial in origin, and it is often met with in arteriosclerosis and myocardial disease. Minor degrees of this irregularity may resemble the youthful type; the severer forms are often complicated by extrasystoles. The beneficial effects of digitalis in these latter forms are owing to the fact that this drug, by depressing the function of conductivity in the auriculoventricular bundle, causes a dropping out of the extrasystoles. Perpetual arrhythmia may appear and disappear either suddenly or gradually; in the latter case we often see a reappearance and steady increase in the size of the auricular wave.

Another variety of arrhythmia which has lately come into prominence as a pathological entity of much clinical importance is:

4. Heart Block. This class of arrhythmias consists of those cases which are due to depression of conductivity (heart block), a condition which may exist in varying degree. It may be manifested by a dropping out of the ventricular beat or by a complete dissociation of auriculoventricular rhythm. The most marked and typical cases are those of the Adams-Stokes syndrome, in which the auricles beat more rapidly than the ventricles. It has been shown that at least the majority of these cases are due to a lesion in the bundle of His. If a sufficient number of muscular fibers in this bundle are destroyed to prevent completely the conduction of the stimulus from the auricle to the ventricle, the latter chamber initiates its own rhythm, beats independently, and a complete heart block is said to exist. If, on the other hand, every second, third, or fourth auricular contraction is able to set the ventricle in action, the condition is termed an incomplete block. Depression of conductivity sometimes occurs temporarily in the convalescent stage of diphtheria, typhoid and rheumatic fever. According to Huebner, the bradycardia, occurring after typhoid and other infections is the result of a toxic depression of conductivity. Heart block may also be produced

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by digitalis, and when already present the administration of this drug still further depresses the function of conductivity, and is therefore absolutely contra-indicated. This effect of digitalis is not necessarily the result of vagus stimulation, for the auricular rate may be unaffected, although the ventricular beats fail; and the pneumogastric nerve seems to act on the latter chamber only through the former. It has been shown that the pneumogastric nerve can produce not only chronotropic, but also dromotropic and inotropic effects (Muskens, Hoffmann, Engelmann). Digitalis seems to exert a specific local action on the function of conductivity.

The existence of delayed conductivity can only be determined by the coincident tracing of an arterial and a venous pulse (usually the radial and the jugular are employed). In normal cases the *a* to *c* interval, as it is called—that is, the time which elapses between the systole of the right auricle and that of the left ventricle—should be not more than 0.2 second with a pulse rate of 70.

If this time is much increased, conductivity is impaired. If the block is complete, the auricular waves in the jugular and the carotid waves bear no constant relations to each other. Experimentation and clinical observation have shown that depression of conductivity may occur as a result of local or general anemia, of toxemia, of degeneration of the cardiac muscle, especially fibroid degeneration and myocarditis of the auriculoventricular groove. Fatigue may be a precipitating factor. I have taken tracings of a case which showed a partial block after exertion which disappeared after a few minutes' rest in the recumbent posture.

Lommel has already called attention to intermittent vagus activity resulting from fatigue.

From a clinical standpoint it is important to decide whether a heart block is nervous or muscular in origin, since the former is much more amenable to treatment, and of less serious prognostic import. In attempting to determine this question we must not forget that heart block may be brought about by secondary dromotropic effect of other disturbances, such as a high pulse rate, which does not allow sufficient time for a restoration of functional activity.

In the heart block due to muscular causes the pulse rate is generally

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lower than in the nervous form. In the former instance the pulse is regularly intermittent, in the latter variably so, and much influenced by respiration. If the administration of a physiological dose of atropine has no effect upon the heart block, the vagus factor has been eliminated.

Heart block may occur between any two parts of the cardiac musculature possessing spontaneous rhythmicity if the bridge of connection be severed or the passage of stimulus be interfered with.

Some tracings published by Gibson seem to indicate that heart block may also be caused by depression of excitability.

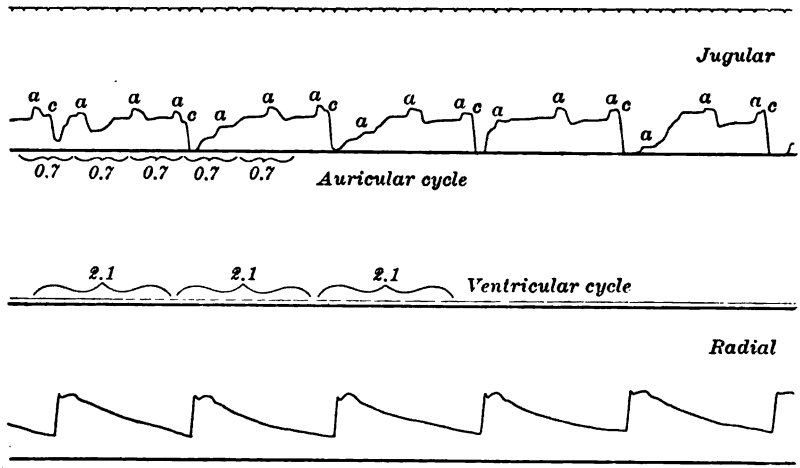

FIG. 9.—Adams-Stokes disease, with a three-to-one rhythm. There are three contractions of the auricle to one of the ventricle.

Fig. 9 was taken from a case of Adams-Stokes disease. It represents an incomplete heart block with a three-to-one rhythm. The ventricle responds only to every third auricular stimulus. The function of auriculoventricular conductivity is much depressed, the *a* to *c* interval lasting four-tenths instead of two-tenths of a second. Death occurred about three months after the first attack of vertigo.[1]

Fig. 10 is a tracing from another case of Adams-Stokes disease, occurring in a man of middle age, who has been under observation

[1] Case referred by Dr. J. W. Irwin. A full account, with histological examination, will soon be published.

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for three years, at which time he was admitted to the Philadelphia General Hospital complaining of dyspnœa on exertion. Tracings made three years ago show the same kind and degree of heart block, but since that time he has been unable to work, and vertigo as well as syncope have been added to his list of symptoms.[1] At the time this tracing was taken his systolic pressure was 145 mm., the diastolic 80 mm. The radial pulse was 30 per minute, both erect and recumbent. It was unaffected by respiration, exercise, or the administration of atropine.

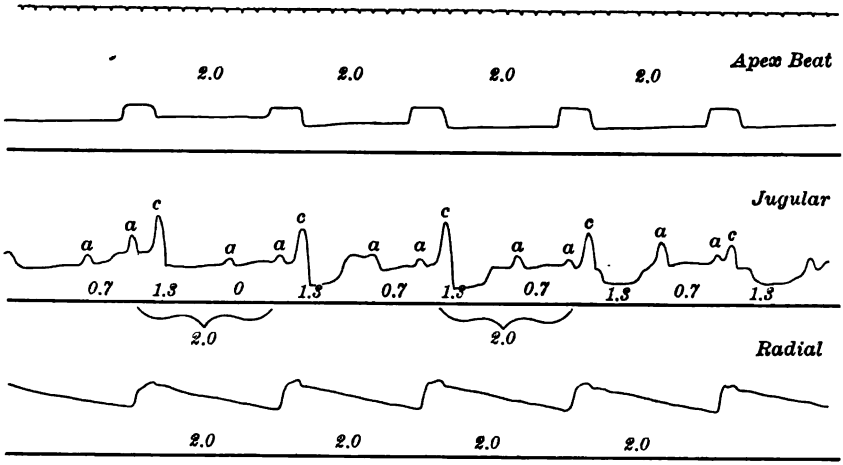

FIG. 10.—Adams-Stokes disease, with a two-to-one rhythm.

Fig. 11 is from another case of heart block with a two-to-one rhythm occurring in a man, aged fifty-eight years, who complained of dyspnœa on exertion, precordial pain radiating to the arms and head (occiput), with vertigo of slight degree. He was the subject of advanced arterial degeneration and had marked hypertension. The highest figures recorded were 235 mm. for the systolic and 140 mm. for the diastolic pressure. At the time this tracing was taken the pressures were 150 mm. and 100 mm. respectively while recumbent, and 175 mm. and 110 mm. while erect. The radial pulse erect was 70; recumbent, 54 per minute.

[1] I am indebted to Dr. F. B. Stahl for the opportunity of making the second series of tracings from this case.

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It would seem, therefore, that we were dealing with a case of heart block in the early stages, since the pulse rate was still affected by change of posture. On auscultating over the body of the heart, two clear, muscular sounds were heard in addition to the aortic second sound, which was high-pitched, clear, and ringing, during which time only one pulse beat appeared at the wrist. Furthermore, every other auricular contraction was followed by a ventricular response. The radial pulse shows the characteristics of arterial hypertension.

Fig. 12[1] is a tracing taken from a man of about fifty years of age, a business man of the modern "hustler" type, in June, 1907. He complained of some dyspnœa on exertion, and an examination of

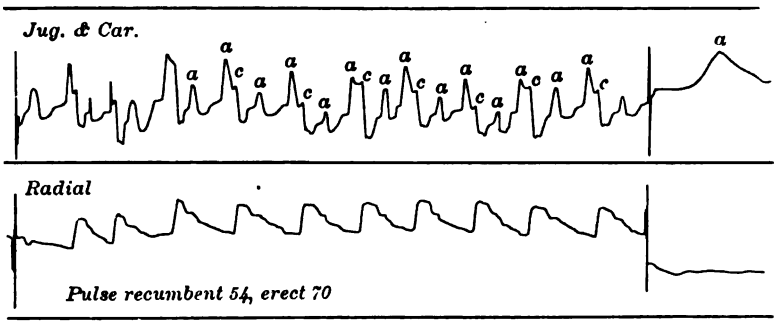

FIG. 11.—Auriculoventricular heart block, occurring in a case of arteriosclerosis with marked hypertension and symptoms of angina pectoris. Two-to-one rhythm.

the chest revealed the physical signs of aortic insufficiency, mitral insufficiency, and a dilated hypertrophy. He had lost twenty-five pounds in weight within one year, and had taken three series of Nauheim baths in this country. The radial tracing shows the typical large-waved, collapsing pulse of aortic insufficiency, with rather well-marked dicrotic wave, perhaps the result of the mitral insufficiency. He had no fever. The jugular wave is of relatively normal proportions, and shows no evidence of auriculoventricular dissociation. Every auricular wave is followed by a ventricular wave. The blood pressure was 140 mm. systolic, 90 mm. diastolic. During the summer his pulse rate fell to as low as 37 per minute,

[1] Case referred to me by Dr. J. H. Musser, as was also the following case.

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and for a period of six weeks remained between 37 and 40, gradually getting up to 60. During this time he felt restless and weak, but had

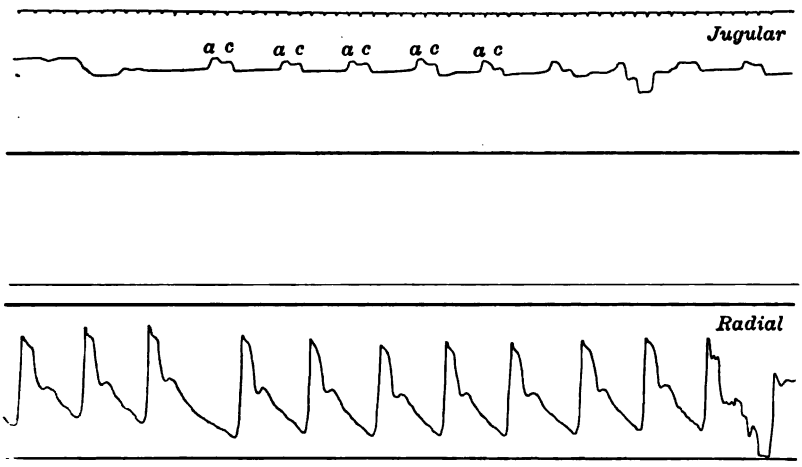

FIG. 12.—Aortic insufficiency and mitral insufficiency. Normal coördination of the auricles and ventricles is shown by the jugular pulse.

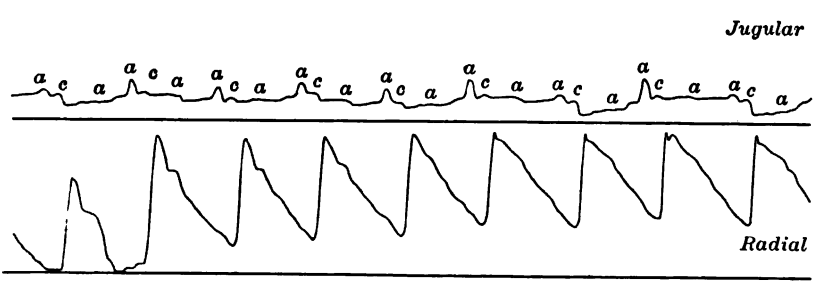

FIG. 13.—The same case as Fig. 12, taken five months later. Distinct heart block with a two-to-one rhythm.

no vertigo or syncope. On resuming his active career he found that when physically tired his pulse would beat at 40, but that rest in the recumbent posture would, in a short time, restore the rate of 60.

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In November, 1907, I had another opportunity of making a pulse tracing (Fig. 13).

Here we are dealing with a distinct case of incomplete heart block, in which the rhythm varies between two-to-one and three-to-one. The pulse rate was 48 per minute, and shortly after the preceding tracing was taken, the patient having been in the recumbent posture, returned to 60 with a disappearance of the heart block, as is shown in Fig. 14.

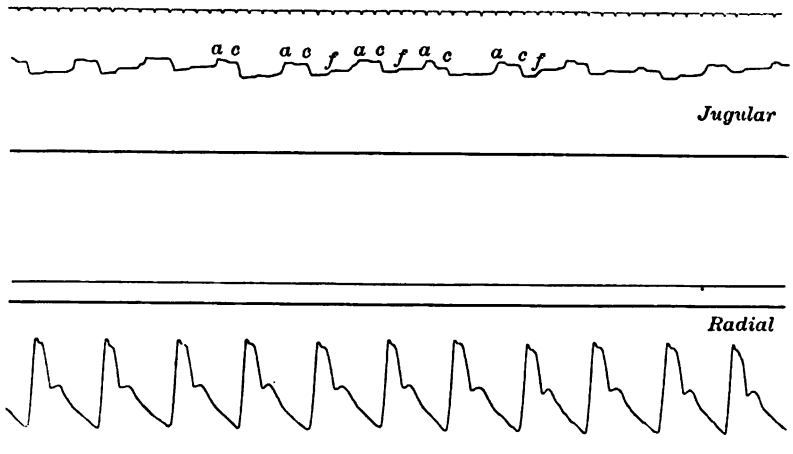

FIG. 14.—The same case as the two preceding figures, after rest. The heart block has disappeared, and the normal auriculoventricular rhythm has been restored.

The last two cases described are, I believe, the first instances recorded of what with strong probability appears to be the early developmental stages of Adams-Stokes disease. How long patients afflicted with this malady may live probably varies greatly under different circumstances, being dependent of course on the extent of damage done to the auriculoventricular bundle, and the character of the lesion affecting it. The case from which tracing No. 5 was taken was known to have had heart block three years ago, and doubtless had had the condition for some time before he came under observation, since his lesion has been very slow in progressing. It is more than probable that careful study by cardiosphygmographic means of many cardiopaths will teach us that heart block

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is a much commoner lesion than we have heretofore been led to suppose. In fact, when we consider the anatomical position of the bundle of His, it is rather surprising that it is not more frequently damaged by inflammatory processes in its neighborhood.

Fig. 15 illustrates another case of heart block due to the administration of digitalis. In this instance the pulse rate dropped suddenly from 80 per minute to about 40. At the time this tracing was taken it was 38.[1]

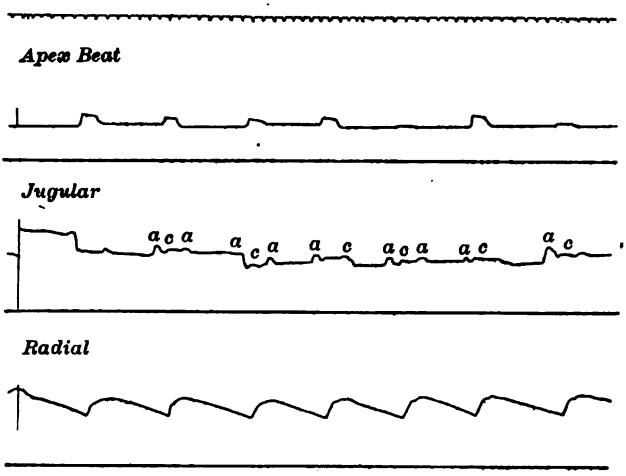

FIG. 15.— Heart block precipitated by the administration of digitalis.

The last form of arrhythmia remaining to be considered is the rarest variety, namely:

5. DEPRESSION OF CONTRACTILITY—PULSUS ALTERNANS. In this class of arrhythmias are placed the cases which are inotropic in origin; that is, those due to depression of contractility. Contractility is easily measured, since cardiac contractions are always maximal if the stimulus provokes any contraction. They are characterized by the alternate appearance on the arterial sphygmogram of a large and a small wave, either with or without increased pulse rate. True cases of this condition are not the result of extrasystole, since they are not followed by compensatory pauses, nor are they due to depres-

[1] I am indebted to Dr. A. O. J. Kelly for the opportunity of studying this case.

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sion of conductivity, as can be determined from the jugular tracing; they arise from heart weakness. The heart while at rest may contract with regular force, but when increased effort is called for uniform contractions cannot be produced. The contraction is slowly accomplished and the muscle insufficiently rested by the time the next stimulus arrives. Hering has described as pulsus pseudo-alternans those cases which upon analysis prove to be the results of extrasystoles. Cases in which the smaller wave occurs too soon are extrasystolic in origin; if the diastolic period is normal, it may or may not have this origin, but if the small wave is retarded we have to deal with a true pulsus alternans. This form of arrhythmia is explained by Wenkebach as follows: With diminished contractile power, if a single normal stimulus happens to occur a trifle too early, it finds the myocardium in a still more hypodynamic condition than did the previous stimulus. As a result, the succeeding contraction will not only be smaller, but will, as has been shown by Hoffmann, be performed more rapidly. If, then, the next stimulus does not occur too late, the longer pause will permit a more perfect restoration of function, and the next wave will again be large. This, in turn, being of longer duration and its pause therefore shortened, will be once more succeeded by a smaller wave.

Clinically, pulsus alternans is recognized by this peculiar, regular alteration of large and small beats. Characteristic of it is the fact that once established it persists for long periods of time, being thus in marked contradistinction to the cases of extrasystole which are irregularly intermittent, as a rule, and have never been known to occur steadily and regularly for weeks. Furthermore, extrasystoles when present tend to vary their appearance during different phases of the diastolic period. The difference in the size of the two waves of a pulsus alternans may vary within wide limits; generally we may assume that the amount of cardiac weakness is directly proportionate to the smallness of the second wave. It also often bears a direct relation to the rapidity of the rate—the more rapid the action the greater the alteration. Pulsus alternans is met with in severe myocardial and valvular lesions. It is a distinctly rare condition. Wenkebach states that he has encountered but two cases. The

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condition is met with in the arterial hypertension of nephritis and angina pectoris, but it sometimes occurs after infections (influenza), and in such cases may disappear.

Thus far we are in possession of but little knowledge concerning stimulus production and tonicity. Gaskell has shown that the latter property may be altered by various drugs, and it has been suggested that cardiac dilatation may result from depression of this function.

PROGNOSIS. It is sometimes difficult to draw definite conclusions as to the prognostic importance of an arrhythmia. Of course, the age of the individual, his occupation, habits, and social condition, have to be taken into consideration. We can also obtain much valuable information from the patient's general condition, the state of the bloodvessels, arterial tension, pulse rate, etc., and especially from the effect of exertion upon these factors. We can rarely afford to prophesy on the basis of the sphygmograph alone any more than we can on the clinical thermometer, and yet both of these instruments are useful adjuvants in diagnosis.

Arrhythmia of muscular origin is more serious than that due to nervous causes. As has already been pointed out, we can determine the presence of the latter by deep breathing and by the effect of the atropine test. Further, the time and cause of an arrhythmia will much modify our opinion of its seriousness. Thus, for example, extrasystoles occurring during the convalescence from infections have generally very little importance, whereas the appearance of this symptom during the fastigium is often of sinister import.

The respiratory type of irregularity is generally of trivial consequence. Extrasystoles, although often insignificant, require careful investigation, especially if occurring in middle-aged or elderly people. Perpetual arrhythmia is always serious, and the same may be said of that rare condition, pulsus alternans. Auriculoventricular heart block also is unfavorable, for although in some subjects the attacks may be precipitated by the injudicious administration of digitalis and disappear after withdrawal of the drug, and other subjects may live for a number of years despite its presence, the symptom always indicates a disturbance of muscular functionation.

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DISCUSSION.

DR. ALISON SCOTT: I do not consider myself competent to discuss this paper, but I do wish to thank Dr. Norris very much for the excellent work he has done on this subject. Those of us who are practising internal medicine are much interested in research of this character, and we all feel that we are not competent nowadays to make definite statements about hearts unless we have made careful tracings from the jugular, the apex, and radial pulse. As Dr. Norris has demonstrated tonight, the administration of remedies we are accustomed to use in practically all cases of cardiac insufficiency seem to precipitate difficulties.

DR. DAVID RIESMAN: Up to within a few years ago, it was thought that the clinical pathology of the heart was a closed chapter; but the researches of which Dr. Norris has spoken show that there is a great deal more to learn about the heart; that in some respects we are only on the threshold of exact knowledge. Dr. Norris' demonstration has been very clear and satisfactory. The point of which I wish to speak is the similarity that at times exists between Adams-Stokes disease and petit mal. Some time ago I saw a young man, aged twenty-two years, who complained of fainting spells and attacks of vertigo; they would overtake him on the street. My first impression was that it was a case of petit mal, and I prescribed the usual remedies—the bromides, in fairly large doses—but without any benefit. When I saw him again, I found that he had a pulse of 48, which led me to think of the possibility that the man had a form of Adams-Stokes disease, perhaps a functional type of that malady. I prescribed tincture of belladonna, and the attacks promptly ceased. At the present time I have under observation a woman who has the Adams-Stokes syndrome for the third time. She is about seventy years of age, arteriosclerotic, and has a dilated heart without any murmur. Two years ago she had attacks of unconsciousness, with slow pulse and slow respiration. The attacks lasted only a moment, but recurred frequently throughout the day for many days in succession; they finally subsided following the use of atropine and nitroglycerin. She had another attack last October, and the third began during the past week. I have seen her in two attacks occurring within a few minutes of each other. I sat down by her bed, and on putting my fingers on her wrist was horrified to find she had no pulse; after ten seconds it reappeared. In the other attack, which was exactly the same, I listened to her heart while I kept my fingers upon the radial artery—there was no pulse whatever, but over the heart I counted 180 feeble pulsations. In an instant the heart beat dropped to 112, becoming stronger and louder, and the pulse returned at the wrist. The interval, of what I might term complete asphygmia, lasted twelve seconds; during that time, the patient lay with glazed and staring eyes. For the last few days there have been no attacks. The patient is taking nitroglycerin and atropine.

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DR. GEORGE W. NORRIS: In reply to Dr. Riesman's question, there undoubtedly may be a functional form of heart block. I think one of the cases I reported tonight, which occurred in an individual when tired, and disappeared after he had rested, showed that the functional activity was capable of wide variation, fatigue depressing activity so that it got below normal. It has also been reported after infectious diseases.

As to the resemblance of Stokes-Adams disease to petit mal, I can readily see how that might occur, but no case of that kind has ever come under my observation. A. E. Russell has suggested that cerebral anemia, presumably due to arrest of heart action just prior to the epileptic seizure, may be a contributing factor in the production of unconsciousness, but Munson's tracings taken before and during epileptic convulsions showed only acceleration of rate—neither irregularity nor arrest of the beat—occurred.

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THE LOCAL USE OF MAGNESIUM SULPHATE SOLUTION IN THE TREATMENT OF ERYSIPELAS, WITH REPORT OF CASES.[1]

By HENRY TUCKER, M.D.,
SURGEON TO THE GENITO-URINARY DEPARTMENT OF THE PHILADELPHIA GENERAL HOSPITAL.

On account of the uniformly good results obtained in my wards at the Philadelphia General Hospital in all varieties of inflammation treated by this method, I was enabled through the courtesy of Dr. Joseph S. Neff, Director, to apply this plan of treatment to the erysipelas cases.

The following series that are reported in full were either of an exceptionally severe infection, or were complicated by some grave condition; the remaining cases were uncomplicated and are merely given as a matter of record.

Case I.—J. Q. Admitted on the fifth day of the disease. Incontinence of urine and feces; delirious; face violaceous; covered with crusts; purulent conjunctivitis. Swelling subsided and all symptoms relieved in three days.

Case II.—F. S., aged three years. Erysipelas of cheek and gonorrheal vaginitis; temperature, 101°; child relieved in twenty-four hours; temperature normal; all local symptoms disappeared at the end of the third day.

Case III.—R. R. Erysipelas of the face complicating a scalp wound; the wound dressed antiseptically and the solution applied to the face; all local signs cleared up within thirty-six hours.

Case IV.—S. S. Admitted delirious; history of anuria for two days: Facial erysipelas; scalp also involved; diffuse bronchitis; acute parenchymatous nephritis. Given diuretic lemonade enteroclysis; hot stupes over the region of the kidneys; magnesium solution applied. Two days later patient markedly

[1] Read May 6, 1908.

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better, mind clear, a fair quantity of urine being voided and local color beginning to fade; the following day temperature dropped to normal; local symptoms entirely relieved.

CASE V.—M. S., aged fifty years. Erysipelas of the face and scalp; glands of the neck swollen and tender; face and ears covered with crusts; lungs congested at bases; acute desquamative nephritis; case incontinent, delirious. Fourth day: mind clear, desquamation beginning; temperature normal. Sixth day: face again became red; urine diminished; temperature rose to 104°. Mask reapplied; the following day all symptoms again subsided; urine increased in amount; kept in bed several days on account of nephritis.

CASE VI.—T. S., aged twenty years. Facial erysipelas; acute bronchitis; urine loaded with albumin and casts; glands of the neck greatly swollen; ears distended with serum; numerous blebs on face; violently delirious; incontinence of urine and feces; temperature 104°. Three days after admission, mind clear, tongue clean and moist; local redness subsided; temperature normal.

CASE VII.—E. S. Erysipelas and delirium tremens; erysipelatous condition cleared up in two days, but the redness faded slowly. The mind clear the fifth day after admission.

CASE VIII.—J. A., negro. Erysipelas involving right side of face and ear, sharply demarcated; mitral disease with hypertrophy complicated this case. Temperature 106° on admission; pain relieved in twenty-four hours; patient comfortable; lesion did not extend; full recovery at end of the third day.

CASE IX.—E. C., aged sixty years (female). Facial erysipelas and advanced myocarditis; ill two days prior to admission. Temperature normal and all local symptoms relieved in twenty-four hours.

CASE X.—M. Mc. Erysipelas following contusion of the nose. The following day all constitutional and local symptoms had abated; third day fully recovered.

CASE XI.—J. W. Entire face involved, resulting from an injury during a debauch; the following day all local lesion disappeared rapidly; pain entirely relieved; discharged cured on the fourth day.

CASE XII.—R. D. Erysipelas of the face and forearm. The infection commenced on the left forearm; two days later the face became involved. On admission both eyes tightly closed. The day following, comfortable and able to open eyes; two days later the local condition approaching normal. Discharged on the fifth day.

CASE XIII.—J. O. N. Condition the same as in Case XI; entirely well on the fourth day.

CASE XIV.—J. K. Admitted with slight pain; swelling and redness of both legs, extending rapidly from ankles to knees. The following day all local symptoms had disappeared.

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CASE XV.—F. S. Transferred from the medical ward. History: pneumonia and pleurisy with effusion. Temperature 103°. Scalp, neck, left cheek, and eye involved. Fourth day: process limited; area less bright in color and margin not so sharply defined. Sixth day: resolution well advanced. Eighth day: well.

CASE XVI.—R. R. Malignant growth of the lower jaw complicated by extensive erysipelas; the erysipelatous condition cleared up on the third day, but the patient died from exhaustion on the seventh day after admission.

CASE XVII.—M. McC. Transferred from the medical ward. Chronic interstitial nephritis; aortic and mitral disease; marked edema of the legs and back. Erysipelas of the left leg and thigh and anterior surface of the right leg; the next day the affected area had lost its bright-red color; swelling less; edges not so well defined. On the third the patient became very much weaker and died.

CASE XVIII.—A. K., aged sixty-seven years. Chronic interstitial nephritis; arteriosclerosis; myocarditis complicated by facial erysipelas; marked dyspnea. The case did not improve and patient died on the fourth day.

CASE XIX.—M. S., aged sixteen months. Face and anterior half of the scalp involved. This case is reported in detail to show the rapid reduction of temperature and pulse that frequently occurs. Admitted 3.15 P.M., axillary temperature 104°, pulse 140; 5 P.M., axillary temperature 101°, pulse 130; 8 P.M., axillary temperature 101°, pulse 110; 11 P.M., axillary temperature 98°, pulse 110. Temperature did not exceed 99° after the first day; entirely well on the fifth day.

In thirty-five uncomplicated though severe cases all recovered within from two to seven days; pain and the usual local discomfort were relieved in a few hours, no internal or so-called specific treatment being given. In the entire number of cases above quoted internal treatment was not used unless urgently indicated by some complication, so the local application must have the credit for the results obtained.

TECHNIQUE. The application consists of a saturated solution of magnesium sulphate in water. This is applied in facial cases on a mask consisting of from fifteen to twenty thicknesses of ordinary gauze, of sufficient size to extend well beyond the area involved, a small opening being made to permit breathing; no opening, however, is cut for the eyes. The mask is then thoroughly saturated with the solution, applied and covered with oiled silk or wax paper, and wet as often as necessary to assure a moist dressing, usually

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once in two hours, depending on the time of the year, or the temperature of the room. The dressing should not be removed oftener than once in twelve hours, to permit inspection of the parts, and then immediately reapplied; the affected area should not be washed while the treatment is employed. The advantages of this form of treatment over others are as follows:

1. The drug can be obtained in any country store, is easily made into solution, is inexpensive, non-toxic and clean, also easy of application if the directions are properly followed.

2. The patient very promptly obtains relief from the distressing local symptoms usually present.

3. The temperature rapidly falls to normal, usually during the second twenty-four hours, and does not rise again, thereby eliminating possible complications from fever.

4. Internal medication is not indicated in uncomplicated cases, the only treatment being a milk diet for the first few days, or, to be more accurate, until the temperature reaches normal.

How the results are accomplished, I must frankly admit, I do not know; but having used this method in over 700 cases of various forms of inflammation with uniformly good results, I feel justified in considering it the best local treatment in any variety of inflammation, especially as the greater number of cases treated in the hospital represent absolutely the worst class, from a physical standpoint, belonging to the lowest strata of humanity; poorly fed, anemic, living normally under the worst hygienic surroundings, and many of them users of alcohol to excess. The results obtained in private practice should be uniformly successful.

In conclusion, I wish to express my thanks to Drs. King and Peterson, internes in the hospital, for the interest displayed in this work.

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DISCUSSION.

DR. H. A. HARE: The extraordinary results which have been obtained by Dr. Tucker seem almost beyond belief, and had I not seen these results in my own practice and in the practice of a number of men who have been kind enough to call me in consultation, I do not think that even my confidence in Dr. Tucker would have convinced me of the correctness of his report. The application of ichthyol, which I have followed very sedulously for the last fifteen years, has always seemed to me the best form of treatment. Dr. Tucker's treatment has left ichthyol as far behind as quinine leaves arsenic far behind in the treatment of malarial fever. Like Dr. Tucker, I am free to confess I have no explanation of these extraordinary results. The effect which is produced on the erysipelas after the magnesium sulphate is almost like the effect of a hot sun on a snowball, the swelling can often be seen to go down in a very few hours. Only last night I saw a case in consultation at Wenonah, N. J., and asked the attending physician as to the results in a severe case of facial erysipelas I had seen with him some weeks before. In that case we had ichthyol on one side of the face and the magnesium sulphate on the other. It was ludicrous to compare the results; the disease ran its course on the ichthyol side of the face, with about the ordinary diminution in the patient's suffering, but the magnesium sulphate side, the pain diminished at once, and the swelling did not spread at all. In another case I know of the magnesium sulphate after the first hour, instead of relieving pain, produced intense scalding or burning pain, reminding one of the old description of erysipelas as St. Anthony's fire; then the brawny line of demarcation disappeared and the redness and pain also disappeared.

It is also interesting to note, because even more inexplicable, the effect these applications have on inflammations in other parts of the body, for example, in the treatment of orchitis, about which Dr. Tucker did not speak, I think, and about which Dr. Belfield, one of the most prominent genito-urinary surgeons of the West, has spoken in terms of praise. How wrapping up the scrotum in a saturated solution of magnesium sulphate can within a few hours affect an orchitis or an epididymitis which has no direct connection with the anatomical or circulatory centres I cannot understand, but it does. So, too, acting under Dr. Tucker's suggestions, I have treated violent forms of pain with no apparent inflammation, with magnesium sulphate, with astonishing good results. About a year ago I had under my care a man with locomotor ataxia. He had attacks of frightful pain in the ball of his foot, coming on in crises at night. Just to see what effect I would get, I wrapped the foot in a saturated solution of magnesium sulphate. On questioning him the next day, he said the pain was gone in a remarkably short time, but he thought the effect left was almost as bad, in other words, the magnesium sulphate produced absolute local anesthesia. In another case, that of a woman sent to my office, a casement window mashed

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her finger, the nail was split and the finger very painful. Just to see what this salt would do I wrapped it up in the magnesium sulphate solution and the result was immediate relief of pain. There is a lesson to be found in this use of magnesium sulphate empirically.

I suppose there is no Fellow of the College who does not prescribe remedies basing his prescription on empiricism—something which is properly but perhaps inordinately condemned—I don't think anyone can accuse me of neglect of pharmacology, for I worked in it exclusively for a number of years. This use of magnesium sulphate illustrates the fact that we often get an effect although we cannot explain how we get it.

Dr. John B. Roberts: Surgeons have learned a great deal if it can be established that magnesium sulphate really has this extraordinary effect in old-fashioned erysipelas, such as they used to see twenty years ago, but do not often see now, because of the care with which wounds are treated. I think possibly Dr. Tucker and others are a little over enthusiastic. In various surgical conditions, such as wounds and complicated fractures, we may have inflammatory conditions which are so much like old-fashioned erysipelas and yet less serious that we are very doubtful whether we should send the patients to the isolation wards of the hospital or whether we should wait a day or so for further development. These cases quickly recover with moist dressings; and it is a question whether the infection was really an infection with the streptococcus of erysipelas or some other surgical condition. I cannot help thinking that some of these cases are not true erysipelas, although they may be mild cases of infection of that nature. The erysipelas, if it be such, disappears quickly under moist dressings and restriction of drying with a covering of waxed paper or oiled silk. May not the rapid subsidence of swelling, the rapid relief of pain in the crushed finger and mumps be due to the mildness of the infection rather than to the specific influence of magnesium sulphate?

Dr. Tucker has given us a very instructive and interesting paper, but I am a little doubtful as to whether we shall find saturated magnesium sulphate solution so valuable in true erysipelas, such as we formerly dealt with quite frequently. May not some of the cases reported be infections with some germ of less virulence than the erysipelas streptococcus? I hesitate to criticise the diagnosis of the instances reported, but I am obliged to confess that I have not been convinced that all of these cases were true erysipelas.

Dr. Hare: As I came into medicine at the time when antiseptic surgery had not yet interfered with old-fashioned erysipelas, to which Dr. Roberts referred, I saw a number of cases of old-fashioned erysipelas and a number of cases of phlegmonous erysipelas. I may be wrong, but I think I know a case of erysipelas when I see it, the distinct line of demarcation, blebs on the ears, and the puffed-up face is erysipelas or I am blind. There is no question that ordinary moist dressings do not do it. I was just as doubtful as Dr. Roberts; I had to be convinced. One case will convince you. I hope Dr. Roberts will try it.

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DR. B. FRANKLIN ROYER: About a year ago, after reading Dr. Tucker's paper upon the use of magnesium sulphate in epididymitis and orchitis, I tried it in the acute glandular swellings in the scarlet fever wards of the Municipal Hospital. It is the best remedy I have used in these conditions, and gives even better results in the periglandular inflammations of scarlet fever and in cellulitis of the neck in this disease. I am not in the habit of applying it exactly as Dr. Tucker does. I use a number of layers of gauze saturated with the magnesium sulphate solution and cover the gauze with oiled silk. I apply an ice bag on the outside and change the gauze often enough to keep it moist. The results secured with magnesium sulphate are far better than I have ever had in these conditions by any other method.

I have treated but a single case of facial erysipelas exclusively by this method, and in it the result was most gratifying.

DR. JOHN B. SHOBER: I would like to ask Dr. Tucker whether the 700 cases spoken of were cases of true erysipelas or cases of all the diseases he has treated with magnesium sulphate? I have seen a good deal of surgery in the last twenty years, and I am astounded to hear that any one man has seen 700 cases of erysipelas in Philadelphia in the last eight or ten years. I have talked to many of my friends during the last winter in regard to erysipelas, because one of our distinguished citizens has recently gone through a severe attack. I have met with but two men who in private or hospital practice have seen more than a very few cases of erysipelas. I am very much interested in this paper, but at the same time I am, with Dr. Roberts, somewhat skeptical about it. Might not some other solution do the same thing? For instance, a normal salt solution used in this way or ammonium chloride solution. I remember a good many years ago calling Dr. D. Hayes Agnew in consultation to see with me a case of a man who had fallen on his back and developed a tumor. Dr. Agnew thought a hematoma had formed and advised moist dressings of ammonium chloride solution. In a few days the swelling subsided. I would like to ask whether Dr. Tucker has treated any cases of burns with this solution? It seems to me it might be equally useful in various degrees of burns and scalds.

DR. FRESCOLN: I have had an opportunity during the last two years of following all the cases of erysipelas that have come to the Philadelphia Hospital, treated by the magnesium sulphate method. With the changes of internes in the erysipelas wards, it is interesting to note we have used ichthyol ointment, bichloride solution, Goulard's extract, 5 per cent. sodium salicylate, antiphlogistine, and several other local applications. Lately we have been using this saturated magnesium sulphate solution under Dr. Tucker's suggestion, and I can testify as to the rapidity of the desiccation and the limitation of the erysipelatous condition and also the fall in the alarming constitutional symptoms that we sometimes have. There is another condition in the hospital, namely, mumps, where we find magnesium sulphate useful; we are using it exclusively

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in the isolation ward for mumps and we have a case now in which the parotid glands subside in about one day under this treatment. I might say that in erysipelas I notice we have relapses after the use of these various applications, and it is difficult to tell by just so many days the exact comparative value of each drug. I have also noticed that particularly in the use of magnesium sulphate results in a purulent infection under the lower eyelid. I cannot give statistics, but it seems to occur more frequently with magnesium sulphate; we will get one-half ounce or more of pus from the lid, but magnesium sulphate relieves rapidly the discomfort of the patient and in most cases brings a rapid cure.

Dr. Myer Solis-Cohen: In the medical wards of the Philadelphia Hospital we have used magnesium sulphate for acute articular rheumatism, with very good results. The joints are wrapped in a moist dressing of Epsom salts and covered with oil silk.

Dr. Henry Tucker: In answer to Dr. Shober's question, I have treated approximately over 700 cases of general inflammation. I have used magnesium sulphate in burns with good results.

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A SIMPLE AND ACCURATE METHOD FOR MEASURING THE CLOTTING TIME OF THE BLOOD.[1]

By MYER SOLIS-COHEN, A.B., M.D.,

CONSULTANT PHYSICIAN TO THE HOME FOR CONSUMPTIVES, CHESTNUT HILL; ASSISTANT PHYSICIAN TO THE PHILADELPHIA GENERAL HOSPITAL; PEDIATRIST TO THE JEWISH HOSPITAL DISPENSARY; ASSISTANT PHYSICIAN TO THE DISPENSARY OF THE UNIVERSITY HOSPITAL.

Those who would study the coagulability of the blood are handicapped by the absence of an accurate method for measuring the clotting time.

It is, of course, impossible to imitate outside of the body the conditions that exist within the vessels or in the wound. There are certain recognized sources of error common to all methods for testing the blood. The skin contains substances that accelerate coagulation. Blood leaving a wound contains a slight or great admixture of lymph, which also has the power of decreasing the clotting time. Contact of the blood with a foreign substance causes liberation of the ferment, the amount or rate depending upon the size and character of the contact surface. When the blood, for instance, comes in contact with smooth, clean glass, it clots the more rapidly the greater the extent of the contact. The external temperature has an influence on coagulation, heat hastening and cold retarding it. Currents of air affect the clotting time, which is accelerated by anything causing evaporation and thus diminishing the quantity of water in the blood. These last three factors may be modified more or less by the method of testing employed.

[1] Read May 6, 1908.

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Some sources of error that can always be avoided are dirt, pressure on the part, and (according to some) the production of passive congestion by means of a ligature, all of which shorten the clotting time.

It must be remembered, however, that the coagulability of the blood is supposed to vary in the same individual during the course of the day and on different days.

The apparatus to be used in measuring the rate of coagulation naturally should avoid as many sources of error as possible. Nevertheless, there will always be some unrecognized factors that will influence the result. The personal equation, however, must be absolutely eliminated. An instrument should always give similar results in the hands of different investigators; otherwise comparisons would be impossible. To my mind a simple apparatus—one that can be made and repaired by anyone—is infinitely superior to an expensive instrument that must be procured from abroad and sent there when out of order. Only with such a simple apparatus can the study of the coagulability of the blood become popularized and be at the command of every practitioner.

Many different methods have been employed for measuring the clotting time. As a rule, all are subject to one or more of the chief sources of error, although at the same time they may avoid others.

In two of the best-known methods capillary tubes are used for receiving the blood. The blood is thereby protected from currents of air, but the large extent of glass contact surface has a tendency to accelerate the coagulation. In Vierordt's method the blood in the capillary tubes is tested by pulling a horsehair through the tube and noting whether it is blood-stained, thus adding two more sources of error, an additional foreign body, the horsehair, and the disturbance of the blood by pulling the hair in and out. No attempt is made to regulate the temperature.

Wright solved this last problem by placing the tubes in a tumbler of water at 37° C. (98° F.) or 18.5° C. (65° or 66° F.). He tested the blood by blowing into the tubes at intervals, the blood being regarded as clotted when the column of blood could not be moved.

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My results with this method, as mentioned elsewhere,[1] were very unsatisfactory.

Brodie and Russell experimented with capillary tubes, but soon discarded them. These investigators finally devised an instrument by which a drop of blood is placed in a warmed moist chamber and set in motion by a stream of air, the movement of the corpuscles being observed under the low power of a microscope. The temperature is kept constant by means of a water jacket surrounding the air chamber. Pratt and Grützner simplified the apparatus by eliminating the water jacket and also the glass cone which determined the size of the drop. Boggs in his modification also removed the water jacket but improved the glass cone. These last two methods consequently do not provide against changes in temperature. Some writers regard this of little consequence, but no records are presented supporting such statements. On the other hand, Brodie and Russell found that at 30° C. the blood coagulated in one-third of the time it took to clot when the temperature was 20° C. Wright also found with this method that the blood clotted in two to four minutes at 18.5 ° C. and in one and a half to two minutes at 37° C. The greatest objection to the Brodie-Russell instrument and its modifications, however, is the fact that a current of air is blown on the drop, which undoubtedly mechanically interferes with normal coagulation. The extent of this source of error depends upon the personal factor, as investigators vary in the force and frequency with which they blow upon the drop. The personal equation also effects the determination of the end point, different observers taking various portions of the different stages in the process of clotting as the terminal stage. The Pratt-Grützner instrument has a decided disadvantage in that the size of the drop is not uniform. It has, however, one advantage over the other two models. Being extremely simple, it can be easily made and repaired; the others must come from abroad. Should the air tube become bent, the accuracy of the

[1] Myer Solis-Cohen, A Study of the Coagulability of the Blood in Disease, Univ. of Penna. Med. Bull., 1907, vol. xx, page 56.

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instruments will at once be impaired, as the current of air must just strike the edge of the drop.

Hayem allowed the blood to flow into a flat-bottomed glass test tube and regarded coagulation as having occurred when the tube could be upturned without altering the shape of the clot. A decided disadvantage is the amount of blood required for each test. The rate of coagulation is affected by the number of drops that fall on the side of the tube, which thus provoke more or less rapid precipitation of fibrin. Moreover, it is difficult to appreciate the exact moment that coagulation has terminated.

In another method a drop of blood is placed between two watch glasses, and when it no longer flows according to the movements of the glasses, but adheres to the glass, it is regarded as clotted.

Still another method is to place a drop of blood upon a glass slide, cover it with a coverglass, and watch with the microscope for the appearance of fibrin. Although in these last two methods evaporation by air currents is avoided, the large amount of contact surface seriously interferes with the results. Neither in them nor in Hayem's method is any attempt made to maintain a constant temperature.

The least amount of contact occurs in the methods in which a film of blood is picked up upon a wire ring or loop. The instrument devised by Dr. Biffi, of Lima,[1] consists of a glass rod or tube in which is fused a platinum wire, bent in such fashion as to have a number of loops of equal diameter. These loops are touched to the surface of the drop of blood and then placed in a glass cylinder filled to a certain point with water of a definite temperature. At stated intervals the rod is pushed down so that one loop after another is introduced into the water. When coagulation is complete the loop, instead of becoming perfectly clean, shows a solid red mass.

Goldhorn modified the instrument by increasing the size of the loops and giving them an extra twist.

[1] L. B. Goldhorn, A New and Practical Method for the Determination of the Coagulation of the Blood, Proceedings of New York Path. Soc., 1895, n. s., vol. v, Nos. 1 and 2, p. 9.

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Buckmaster[1] picks up a film of blood on an elliptical metal ring and clamps the shank of the loop in a holder in a moist chamber. Commencing with the film in a horizontal position, he rotates it at intervals into a vertical position and examines it with a magnification of about ten diameters. When the corpuscles no longer fall, leaving a clear space above, but the blood remains of the same opacity throughout, coagulation has occurred. Both these methods have simplicity to recommend them, but little evidence has been presented as to their working in other hands. The chief objection to them is the tendency of such a thin film to evaporate.

The simplest methods of all are those in which the blood is dropped upon a glass slide and studied there. One way is to receive a large drop on a clean slide and from time to time test its consistency with a needle. Or several individual drops of blood of the same size are collected upon a glass slide and at regular intervals a straw of a whisk broom is lightly trailed through each drop in succession until a delicate thread of fibrin is seen clinging to the straw.

Milian allowed a rather large drop of blood to fall upon a glass slide, which at intervals is carefully tipped vertically. The blood is coagulated when the profile of the drop shows a uniform convexity and does not become tear-shaped on tilting the slide.

The above three methods make no allowance for evaporation, changes in the temperature, and variations in the size of the drop. In the first a foreign body is repeatedly introduced into the blood, thus vitiating the results.

Bezançon and Labbé, who used Milian's method, prevented evaporation to some extent by placing a bell jar over the slides, but as they had to expose the blood to the air each time they tilted the slides, the effect of this expedient was only partial.

Hinman and Sladen[2] modified Milian's method by touching the slide to the drop of blood instead of allowing the latter to fall,

[1] Geo. A. Buckmaster, The Morphology of Normal and Pathological Blood, London, 1906.

[2] Frank Hinman and Frank J. Sladen, Measurement of the Coagulation Time of the Blood, and its Application, Bulletin of Johns Hopkins Hospital, June-July, 1907, vol. xviii, Nos. 195 and 196, p. 207.

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and by retaining only those drops that are 4 or 5 mm. in diameter. They wiped off the first drop and counted from the appearance of the second drop. Holding the slide horizontally, they lightly touch its under surface to the drop, and after they have thus picked off several drops they quickly invert the slide to prevent the drops from flowing. Placing a scale under the slide, they measure the drops and wipe off all that are less than 4 mm. or greater than 5 mm. in diameter. In addition to observing the contour of the drop from the side, they were also able to determine the occurrence of coagulation by holding the slide vertically to transmitted light and judging by the changes in density whether there was a flow in the drop. The denser portion is in the centre when coagulation has occurred, and at the lower edge when the drop is still fluid.

A great objection to Hinman and Sladen's method, which they themselves admit, is the exposure to currents of air and consequent evaporation. They go so far as to state that their method is unsatisfactory where the time is very much delayed. But I found the method unsatisfactory often when the blood clotted within normal limits. One drop that measured 5 mm. in diameter had a fluid centre 1.5 mm. in diameter, surrounded by a moderately dry rim 1 mm. wide, the outer rim of the drop being completely dry. The outer border or edge of the drops always evaporated and became hard and dry. Consequently the fluid portion of the drop in the centre which ultimately coagulates is often much smaller than the lowest limit imposed by Hinman and Sladen. Moreover, drops whose bases have the same diameter cannot always be said to be of the same size, for the shallow drop contains less blood than the high drop. The shallow drop also evaporates more quickly than the high drop.

One source of error in Milian's method and in all its modifications is the failure to maintain a constant temperature, which certainly alters the results.

I have devised an apparatus based upon Milian's method of testing the blood, but which eliminates to a considerable extent the errors due to evaporation and changes in temperature. It consists of a German Stender dish, a small pan with an outlet and with a

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bracket for holding a thermometer, a bath thermometer, a wide elastic band, a piece of rubber tubing, a stopcock, and a millimeter scale. The German Stender dish I use is 80 mm. in diameter and 40 mm. high. An ordinary tin baking pan, big enough to hold the Stender dish and the bath thermometer, may be used, an outlet and bracket being made in it by a tinsmith. I paid five cents for a pan and fifteen cents for having it altered. A neater pan with perpendicular sides can be made by a metal worker out of zinc. Space is economized by having the dish oval in shape instead of round. Zinc, not being acted upon by water, is preferred to tin, which rusts, and copper, which turns green. I use an ordinary bath thermometer encased in wood. The rubber tubing is attached to the outlet of the pan and clamped with the stopcock, and the thermometer is slipped into the bracket or holder.

When a test for blood coagulability is to be made, the pan is filled with water of a temperature of 37° C. (98° F.), which is maintained at this temperature by the addition of hot or cold water, any excess being allowed to run out through the rubber tube.

The Stender dish is cleansed, the rim smeared with lanolin or petrolatum, the lid thoroughly cleansed and applied, and held in place by the rubber band. The dish is then placed, everted, in the pan of water.

The ear or finger is next thoroughly cleansed and given a free puncture with a clean, sharp instrument, so that the blood flows freely without undue pressure. The Stender dish is removed from the water, quickly dried, and the elastic band removed. The first drop of blood is wiped off and note is made of the time the second drop appears. This drop while on the finger or ear is lightly touched to the upper surface of the inverted lid of the Stender dish held horizontally. The part is then wiped off and several other drops are similarly deposited upon the lid on either side of the median line, the time of the appearance of each drop being also noted. The dish is now immediately placed upon the lid and the rubber band applied. Placing the millimeter scale under the inverted dish, the size of the different drops is quickly measured and noted. Only those drops are regarded whose diameter

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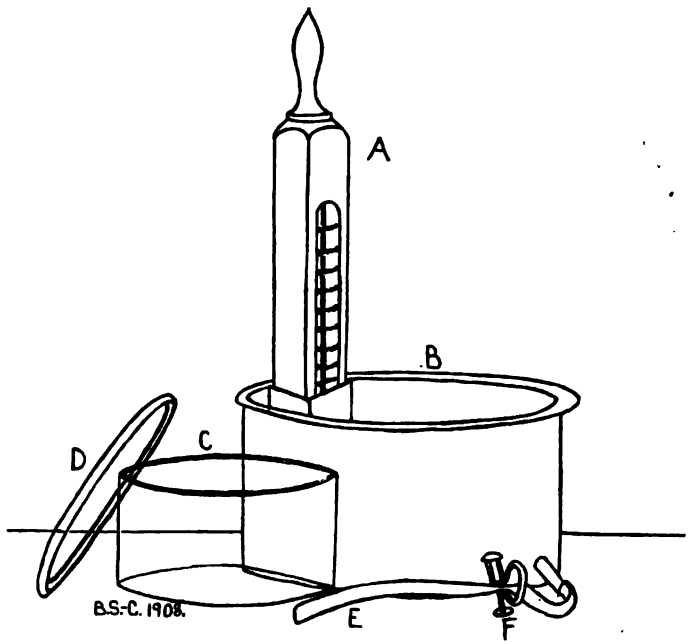
measures 5 mm. or 6 mm. The dish is then placed back in the water bath with the lid underneath.

At intervals the dish is removed from the water and held vertically, while the contour of the drops is rapidly observed from the side or the density studied by looking at the drops through the lid. As quickly as possible the dish is returned to the horizontal position



A, ordinary bath thermometer; *B*, zinc pan (common tin pan will answer); *C*, German Stender dish; *D*, lid of German Stender dish; *E*, rubber tube; *F*, clamp.

and replaced in the water. So long as, when observed from the side, the drops sag, are tear- or pear-shaped, or, when looked at full, are denser below and clearer above, coagulation has not occurred. As soon as the drop is rounded or convex on profile view, and of the same density throughout when seen from the front, coagulation is completed. To confirm this, the dish is removed from the lid and the clot is picked off with a needle or straw. The time at which coagulation was first observed is noted, and the

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period between this and the moment the drop appeared on the part is taken as the clotting time.

With this method results are surprisingly constant. The clotting time of the various drops varies but slightly. Drops of the same size practically all clot in the same period of time. Should there be a slight variation, due to any cause, recognized or unknown, the average clotting time of the several drops will obviate this error.

There is practically no evaporation. A drop when coagulated is of the same size it was when the test was begun.

My experiments thus far make me feel that temperature is an important factor. The test is made with the greatest difficulty at 17.5° C. (65° to 66° F.), owing to the tendency of the drop to run when tilted at this temperature. The clotting time seemed increased at the lower temperature. While wax or paraffin may be used to grease the rim of the dish when the higher temperature is employed, they are inefficient to keep the water out of the dish when the test is made at the lower temperature. Lanolin and vaseline, however, can be used at all temperatures. I prefer lanolin, as it seems to make the lid stick on tighter.

I do not wipe off the drops that are too large or too small, as I desire to limit as much as possible the exposure of the blood to the air. I find it easy to disregard such drops.

Two sources of error are still present in the method just described. The disturbance of the drop by tilting the dish must affect coagulation. Only the diameter of the drop is measured, not its height. This latter factor, however, is probably of minor importance in the absence of evaporation.

Despite these and probably other sources of error which at present I am not able to recognize, the results with the Stender dish method are so constant, especially when compared with a method like Wright's, that I feel encouraged to continue the study of the coagulability of the blood in disease that I began about seven years ago, but which I discontinued on account of the unreliability of the method employed.

The apparatus is so simple that it can be made by any physician, and having no intricacies it cannot get out of order. Its cost is very small. Consequently it is within the reach of all.

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DISCUSSION.

DR. W. E. ROBERTSON: The method appeals to me as being one of the simplest, and apparently one of great practicability. In a series of studies made by Drs. Duncan and Illman and myself—nearly 400 observations at the Episcopal and Samaritan Hospitals—we used particularly Wright's method. In a few instances the old method of Vierordt, which he first suggested in 1878. The Wright method is a rather cumbersome one, in that it necessitates careful cleaning of the tubes each time, and this is difficult, as with blood that coagulates rapidly the capillary tubes become obstructed. So far as the temperature is concerned, we found the lower temperatures are infinitely better. By the higher temperatures coagulation takes place so rapidly—in fifty seconds in some instances—that the margin of error becomes rather great with the Wright instrument. With the 18.5° Centigrade temperature we found that coagulation never occurred in less than one and a half to two minutes. This, of course gives one plenty of opportunity in making a series of observations to minimize the error. Our observations were made with the idea of determining the influence of various factors on coagulation, including drugs alleged to prolong and hasten coagulation.

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OUR PRESENT UNDERSTANDING OF THE NON-TUBERCULOUS (RHEUMATOID) DISEASES OF THE JOINTS.[1]

By JOEL E. GOLDTHWAIT, M.D.,
BOSTON, MASS.

It is not my purpose in that which it is my privilege to present to you tonight to devote much of any time to those features of the rheumatoid problem in which there has been little if any change during the past two years, but rather to take up those features in which the changes are so marked that their presentation may be of interest and possible help to you.

In the first place, we must remember that the disease called rheumatism is not an entity, but that there are many different diseases or conditions which are all classed together under this one head, the same as in times past different diseases were classed together under the term "lung fever," or with the abdominal conditions the term "inflammation of the bowels." A better understanding of pathology has made it possible to differentiate the various disease conditions which were formerly included under these terms. The same change is taking place in our knowledge of the joint conditions and already five or six different disease conditions have been recognized. Of these there is little for me to say to you in regard to the types in which atrophy and hypertrophy of the joint structures are the chief characteristics, as our knowledge of the etiology and pathology has changed but little with these types over that which has already been published.

Of the types in which there has been progress during this period,

[1] Read by invitation, March 4, 1908.

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the first to be considered is the one which has been designated as infectious arthritis. With the cases coming under this head there is comparatively little question in the minds of observers upon both sides of the water as to its infectivity. The only dispute is as to whether one organism or many cause the trouble. One or two men still hold that a specific organism exists, but most of the observers believe that any of the infectious organisms may under certain conditions lead to joint symptoms. The gonococcus, pneumococcus, streptococcus, the typhoid bacillus, the la grippe bacillus, and many others, now known or to be discovered later, under certain conditions apparently result in joint lesions, the special pathological lesion in the individual joint depending upon the special organism, the virulence of the organism, or the degree of the individual's resistance. The pneumococcus in its virulent form is so characteristic in its effect that it can often be recognized before any bacteriological test is made. The same is true of the gonococcus or typhoid bacillus, and with our increasing knowledge the same clinical recognition will probably be possible with many of the other organisms.

Recognizing infection as the cause of these conditions, naturally the obvious principle in the treatment consists first in finding and treating the source of the infection, and it is in this part of the study and treatment of these conditions that very marked advance has been made. Comparatively few of the cases are primarily joint cases, the special joint symptoms being almost always the sequel of some disease condition present elsewhere in the body, exactly the same as the diseases of the ear so often follow "la grippe," or various throat lesions. The primary disease may have been insignificant in comparison to the sequelæ, but this is, of course, no more the case with joint diseases than with other better understood pathological processes. Simple colds, tonsillitis, ulcerations of the teeth, intestinal disturbances, all may lead to joint lesions, and not only is this so, but wherever the joint disease remains active it usually means that some source of active infection is still present within the body. This being the case, it becomes our duty in the treatment of such conditions to hunt out the point of

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infection with the idea of its removal rather than to smother the symptoms with drugs while the primary disease is all the time going on actively. The chronic rheumatism with repeated exacerbations of the acute symptoms usually means just this, and such a case should always be studied with reference to finding the source of the infection.

The first thing necessary is a thorough examination. The throat, the gastro-intestinal tract, the teeth, the genito-urinary tract, should all be examined, as well as cutaneous disturbances from which absorption might result. In the throat in these cases the tonsils are, as a rule, only moderately enlarged, but in them are apt to be certain crypts distended with pus and detritus. If such a source of infection is present, in a case such as is being discussed, it is obviously approaching the subject from the wrong end to treat the joints until the source of the infection has been removed. With the tonsils the accessory sinuses and passages about the throat and nose should always be examined, as in them not infrequently is the trouble found.

The teeth should be examined, with the removal of any possible source of infection.

If disease of the genito-urinary tract is present, from which absorption might occur, this should naturally be corrected, and similarly any other conditions in other parts of the body which could in these ways aggravate the joint lesion should be corrected before we can hope to do much for the joint process. Especially should the gastro-intestinal contents be studied in regard to the organisms present.

A good many times after the most thorough examination which today we are capable of making, we are confronted with the fact that we are dealing with a pathological process, infectious in character, but in which we are unable to locate the source of the infection. This does not mean that these cases are hopeless and that there is nothing to do other than to drug the patient to make him comfortable, but under such conditions we are obliged to fall back upon the principles of hygiene the same as are insisted upon for the control of tuberculosis. With tuberculosis it is an accepted fact that

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good hygiene is the first requisite in the control of the disease, and with other infections we must at times fall back upon these same principles. It matters not as to the organism, whether the tubercle bacillus or something else, the control of the development of the organism is the same, and with this control the improvement with the patient is correspondingly proportionate. If we can find the source of the infection the problem is comparatively easy, but failing in this we must treat the cases with at least the same degree of intelligence as we do tuberculosis. Antitoxins or vaccines as yet have not been sufficiently perfected to be of much practical value, but it is, of course, only a matter of time before such measures will be available.

The next class of the rheumatoid conditions in which there has been distinct advance of late is in those in whom the local joint lesions are due to strain rather than to true joint disease. In order to fully comprehend this class it is necessary to consider the human body as a carefully balanced machine, and to recognize that any departure from this state of balance must result in strain, which if continued leads to symptoms which are frequently spoken of as rheumatism. If the body is held inclined forward the muscles which should hold the body erect are thrown out of balance and soon tire, so that more than the normal amount of work is thrown upon the ligaments, and, as a result, the joints become painful. In such a disturbance of balance not only is the back strained, but the hips, knees, and feet must also be strained, because the weight is thrown upon them differently from that which represents normal poise. Certain parts of the body are more liable to strain than others, and those which would most naturally be affected are those in which the support is obtained chiefly from the muscles and ligaments. The feet, for instance, are held in proper balance by these structures, and anything which in any way weakens their tone naturally results in the lowering of the arch, with an increase of the strain. The various degrees of flatfoot, while not now often treated as rheumatism, nevertheless illustrate the type of disease in which the joint symptoms are due to imperfect balance with strain instead of true disease. With such a condition present in

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the feet it is to be remembered that all of the strain is not confined to the feet, but that the balance is so delicate that the knees, hips, and back must also be strained in varying degrees. Many of the cases of so-called dry or creaking knees are definitely to be explained in this way, and this being the case the cause must naturally be recognized in planning the treatment.

Another part of the body very liable to strain is the pelvis, the joints of which are supported almost entirely by muscles and ligaments, since the vertical or oblique axes of the joints make possible very little support from the bones themselves. In this connection it is necessary to remember that the pelvis is composed of three bones connected with three true joints, in which, with every man, woman, or child, there is motion unless because of some pathological condition or congenital defect. The axis of the pubic joint is vertical, while the axis of the sacro-iliac joints is oblique, allowing play in all directions. The common motion is a tilting of the sacrum upon a transverse axis drawn through the centre of the sacrum, and when tilted backward at the top this naturally increases the pelvic diameters, but narrows them at the outlet, and vice versa—facts which are of importance in connection with obstetrics. In pregnancy there is a physiological relaxation of these joints which can aid or retard labor in proportion to the position in which the sacrum is held. In menstruation there is also a physiological relaxation of these joints, a fact that explains many of the backaches at these times, and offers a reason why at that time the physical labors of a woman should be lessened rather than increased.

Once recognizing that in these joints we have motion and that stability must depend upon the muscles and ligaments, we must recognize in the first place that they are liable to any of the disease conditions which are seen in other joints, and that not only this, but because of their mechanical adjustment, they are more liable than the others to strain. The simplest strain is produced by stooping, and the relief brought about by stretching when the erect attitude is assumed is simply the correction of the position of the sacrum so that the strain is relieved. If such a strain is con-

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tinued, or if an unexpectedly heavy load is undertaken, not infrequently the joints give way and a "stitch in the back" occurs, which is usually simply a sprain of one or both of the sacro-iliac joints. If as the result of the strain or sprain the muscles and ligaments become weakened, a chronic strain results with backache and other symptoms of strain. The discomfort under such circumstances will be felt most when the patient is standing, sitting in the lounging attitude, or when lying down upon the back. The reason for the pain in the two latter positions is that in both, the lumbar curve of the spine is flattened or reversed, and as the sacrum moves with the lumbar spine it is dragged backward at the top, and the sacro-iliac joint strain results. As proof of the condition, the relief of the pain when the back is supported by pillow or otherwise is very striking. The pain in these cases is referred to the lower back or is referred to the legs, the latter being part of the irritation of the sacral plexus by the strain of the joints. It is this that explains many of the cases of lumbago and sciatica.

Wherever the weakness of the pelvic joints exists it must be remembered that as the pelvis represents the structural base of the body if the joints are relaxed the muscles of the trunk and legs cannot be used normally, and strain of the joints of the back and of the legs must result. Many of the cases of marked helplessness start in this way, there being no real disease present, but instead the condition is the result of strain from use of the part with the normal adjustment disturbed. These cases can properly be designated as cases of static joint strain or imperfect poise.

Another condition which is similar in many respects to that just described is that in which peculiarities in the development of the scapulæ exist and result in strain or irritation at the upper part of the spine and scapular region. Normally the scapula is flat and rests upon the ribs, moving about over the ribs and muscles without difficulty. Not infrequently, however, as the result of the imperfect adjustment of the clothing in childhood, the upper part of the scapula bends forward, so that when the shoulders are erect the upper angle of the bone runs over and catches upon the ribs, causing crepitation and pain. The pain is referred either to the point

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of pressure of the scapula upon the ribs or to the distribution of the upper half of the trapezius muscle, which becomes strained in the unconscious effort of this part of the muscle to hold the scapula above the ribs and thus prevent the irritation. This condition is naturally a mechanical one, and while frequently receiving rheumatic drugs, naturally is to be relieved by mechanical measures.

Such, briefly, is the subject of rheumatism or the non-tuberculous joint diseases as we understand the problem today, and in closing, my plea is that you will remember in the first place that rheumatism as a disease is not an entity, and that you will try by careful study to find out the nature of the condition causing the symptoms and apply such treatment as your common sense would prompt after such study, whether or not it be in keeping with conventional methods. Remember that the chronic patient is entitled to the same care and study as the acute case, and that oftentimes more depends with the chronic case upon this care and study than with the patient having some acute and usually self-limited disease.

DISCUSSION.

DR. J. C. WILSON: One thought brought to my mind during the latter portion of Dr. Goldthwait's address is that embodied in the maxim, "Happy is he who is able to know the causes of things." Perhaps my interest and enthusiasm from that point of view is greater than that of some of my colleagues who are engaged in the special work carried on by Dr. Goldthwait and which is largely an unfamiliar one to me. The last portion of his address as well as the first part which related more especially to medical matters interested me very much because I saw that he was breaking down some of the traditions, particularly with reference to the reflex pains of neurasthenia. He has pointed out in a very satisfactory way matters about which the medical man thinks too carelessly. His remarks bearing upon internal medicine are in the highest degree suggestive. I was deeply interested to hear him say that some forms of arthritis could have been so closely associated with disease of the accessory sinuses of the nose and so promptly relieved by operation.

Regarding the forms of arthritis described as rheumatic, it occurs to me that Dr. Goldthwait's discussion does not elucidate with as much clearness as in other matters his views in regard to the typical forms of rheumatic fever characterized by a fugacious variety of acute arthritis, in which from day to day

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one joint after another is involved, not always showing the same degree of disturbance, and here and there a more persistent inflammation. Has he found this form of polyarthritis due to a local infection and promptly curable by the removal of such a focus, or has he not found it to be due to an infection which is generalized from the outset of the attack? Dr. Goldthwait's communication emphasizes the correlation and interdependence of the various fields of medical work and especially the usefulness of special work, scientifically conducted, in cases that are not manageable by internal medicine on the one hand or surgery on the other. He has shown us that our work in the various departments of medicine is so closely associated and interdependent that we cannot, if we aim to achieve the best results, afford to overlook what others are doing in different but contiguous fields of labor.

DR. G. G. DAVIS: I should like to have heard Dr. Goldthwait go farther into the classification of these conditions. From the title of his paper the question immediately suggests itself, "What do we mean by the diseases which are infectious but non-tuberculous?" We know that there are a large number of diseases which, whether actually diseases of the joints or symptomatic, we call diseases of the joints, which it is demanded should be placed in certain definite classes. The tuberculous class has been recognized and cut off from the main division. We might also speak of a traumatic class. For example, a person may injure a joint, and that injury may produce an inflammation which is non-bacteriological in origin. Dr. Goldthwait has alluded to an infectious class, and the question arises, What is an infectious arthritis? Not only has the tubercle been identified, but the infectious feature has been demonstrated in other joint affections. Dr. Goldthwait has referred to the pneumococcic, the typhoid, the gonorrheal, and others, but when you take all those out, you still have a very large number left, perhaps the greater number, and the question is, Where shall we place the diseases which are ordinarily called rheumatoid? We know there is such a thing as an infectious disease. Is there such a thing as trophic disease; a disease, so to speak, of nutrition? Suppose Dr. Goldthwait has alluded to infection coming from the intestinal canal; is that infectious disease? If the microbe crawls from the intestine to the joint, then it is an infectious disease of the joint; but suppose it stayed in the intestine and produces toxins which pass through the blood and so affects the joint, is that infectious disease or trophic disease? The question is, where to draw the line. But I believe the "handwriting on the wall," and that one by one we are gradually identifying an individual cause for each of these joint diseases. Dr. Goldthwait in his sinus case has shown another cause. Pyemia and septicemia cases we know are due practically to pyogenic organisms. Acute articular rheumatism has likewise been placed among the infective diseases. I should like to know where to place rheumatoid arthritis. I say "rheumatoid arthritis" simply for convenience, and ask whether in the gradations from the acutely inflamed smaller joints so characteristic of infectious troubles, up to the slow chronic

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conditions seen in the diseases of the hip, they are infectious or non-infectious? The facts have a vital influence upon treatment. Dr. Goldthwait has shown how in a certain number of cases treatment is directly dependent on our knowledge of the cause, and that as we are able to fathom the cause so we are able to institute intelligent treatment. This question was recently discussed before the British Medical Association, and the gentleman who introduced it suggested that rheumatic affections be diagnosticated by the influences that the salicylates have on them. If you do not know the cause you may institute the wrong treatment. Suppose a certain disease is trophic and not infectious, then to stimulate nutrition would be desirable, and yet I have seen cases kept on the salicylates for long periods of time and they are known to be marked depressants. This shows the prime necessity of an accurate diagnosis. So far as I am concerned, I have nothing new in the way of classification to offer. It is to our present guest that we are indebted very largely, indeed, for the greatest aid which has been given in this question of classification. He has given us a working classification, but I believe the whole question will have to be solved by a patient working out the pathology of the individual cases.

A consideration of the static question shows us the relation of cause and effect and that orthopedic surgery is harking back to hygiene, as medical science and other departments are doing. In other words, when a case of deformity presents itself to the surgeon, he does not simply endeavor to remedy that deformity, but to ascertain the cause. In the static condition it is usually a case of disturbance of the centre of gravity. I am glad that Dr. Goldthwait has presented the subject to us because it shows that surgery is not working along empirically on the plan of the blacksmith, but is endeavoring to find the true causes of deformities and disabilities and to remedy them by appropriate means. Whether that is mechanical or medical is immaterial so long as the cause is discovered and results achieved.

Dr. Charles H. Frazier: The principal reason for accepting an invitation to discuss this paper was a desire on my part to pay some tribute to Dr. Goldthwait's work. There is no one, in this country, at least, who has done more to stimulate interest in this long neglected field. To undertake and pursue lines of investigation in searching for the truth as to the etiology and treatment of these essentially chronic affections required a man of optimistic temperament, and I am sure Dr. Goldthwait is generously endowed with this. I had the good fortune to spend the greater part of a day with him in Boston, and was then greatly impressed with the enthusiastic and optimistic spirit with which he had infused all those, both patients and assistants, with whom he came in contact.

The problems and difficulties of joint surgery is not a development of modern times. If we read the works of Cooper and Brodie, based on observations made a century ago, we find even at that time they realized that the subject had not met with the attention it deserved. Ignorant of the part played by microörganisms, Brodie emphasized the importance of studying the patho-

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logical features and more particularly of the incipient morbid changes in the initial stages of the disease. And so he never lost an opportunity at necropsy to critically examine an affected joint. Later on, but before the days of bacteriology, we find writers hinting at the infectious nature of joint affections. To differentiate them from the rheumatic class which at that time embraced almost all joint affections, they applied the term pseudo-rheumatism.

My interest in this subject was excited primarily by the increasing difficulties that confronted me in the instruction of students. One of the chief difficulties is the confusion as to nomenclature and classification. It is unnecessary for me to repeat the multitudinous terms that have been applied to that form of arthritis commonly called rheumatoid, but recently christened by Hoffa as polyarthritis progressiva, a term which, whatever may be its other advantages, would have but a depressing effect upon a patient already despairing of recovery. Nor need I remind you of the numerous and complicated classifications that have recently appeared, such as the classification of Wright, Nathan, Hoffa, and others. To straighten out the tangle in the minds of the medical profession and to wean them away from that most unfortunate term "rheumatic," a classification should be adopted which is concise and uncomplicated. I believe with others that the only satisfactory classification is one based upon the etiology. With the exception perhaps of the tubercular, atrophic, and hypertrophic types, there is no specific pathological process by which one joint lesion may be differentiated from another. The terminal stage of a traumatic arthritis may not differ from that of inflammatory origin—a gonococcic may differ in no respect from a streptococcic joint. As we have been able to prove, the infectious origin of an increasing number of cases, the mono-arthritic as well as the polyarthritic, this problem is becoming more and more simple. As time goes on, I believe with Dr. Goldthwait that the number of cases of chronic arthritis, heretofore obscure in origin, found to be due to infection will rapidly increase, so that excluding those due to trauma, including static disturbances, those due to such constitutional disturbances as gout and hemophilia, and those due to cord lesions, we will soon be able to assign the great majority of the remainder to the infectious group.

The acknowledgment of the infectious nature of the chronic arthritis opens up at once an intelligent plan of treatment, not only in the attempt to remove the cause, but in the treatment of the lesion itself. The results that have come from studies of gono-arthritis have done much to establish the relation between an infectious source and the joint manifestations. We know now that the Neisser organism may give rise to a polyarthritis of the peripheral joints as well as to the mono-articular type of the disease. The careful search by massage of the prostate for the Neisser organism in cases of seemingly latent infections and the adoption of appropriate treatment have proved, on the one hand, the infectious nature of the disease, and, on the other, the beneficial effect which will follow the treatment of the primary source of infection. We are indebted to Marsh

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for an instructive report containing examples of cases of polyarthritis due to gonococcus, staphylococcus, streptococcus, and mixed infections.

Speaking now chiefly for the infectious group, I believe in the past surgeons have been altogether too conservative in their management of these cases, a conservatism which in many instances is born of fear rather than reason. The present generation of surgeons have inherited from their predecessors a traditional fear of opening joints. The introduction of modern antisepsis and asepsis opened the way for fearless exploration of the cranial and peritoneal cavities, but has not been taken advantage of in the treatment of surgical affections of the joints. The prompt subsidence of the process which attends opening and irrigation of a large joint, for example, in Neisser's infections, should make us consider the propriety of surgical therapy in polyarthritic affections even of the smaller joints.

In the same way, I believe surgeons are too conservative in the management of the chronic forms of arthritis with disability due to callus formation, to joint lipomata, to hypertrophic villi, and to loose bodies. Generally speaking, joint surgery from the operative standpoint has been in large measure neglected except by a few who have made it a special object of study. In hospital practice I find it excessively difficult to interest internes in this class of cases. Most of them still entertain old-time views as to the etiology, and look upon these patients as a class of chronica with little if anything of surgical interest. We are just beginning to appreciate the value of Bier's passive hyperemia in stiff joints. In the introduction of this treatment we have I think one of the most efficient remedies in the treatment of joints, painful and partly ankylosed as a result either of infection or traumatism.

Dr. John B. Roberts: If the Fellows of the College will bear in mind what Dr. Goldthwait has said about pain in the back of people upon whom they operate, the citizens of Philadelphia will owe him a very great debt. The severe pain patients suffer after operation on the ordinary flat glass operating table, or on what is even worse, the table which has a triangular groove to allow the blood to escape under the back of the patient, is preventable by acting on the principles which he has suggested by his researches. A vast deal of suffering could be saved. He has taught us in a sort of casual way, that which I, and perhaps others, have been teaching for some years, namely, that the lumbar pain after operations is often due to the strain on the muscles and ligaments of the back caused by the flattening of the normal lumbar arch when a patient lies on a flat table during a prolonged anesthesia or operation without anesthesia. A sandbag or hard pillow or an inflated rubber cushion should be placed under the loins. Dr. Goldthwait's article on the subject of the movement of the bones of the pelvis, published a year or so ago, delighted me much when I saw that he referred to the subject which I had been considering. When I operate upon an abdominal case and a patient is put upon a flat bed or flat hard table I like to put some support under the lumbar spine unless the patient's buttocks are so slightly developed as not to lift up the lower portion of the spine into the

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curve which is seen usually just above the pelvis. Women who have large buttocks and hips have especial need for such support during operation. It seems to me that many of us are liable to operate without the slightest idea of mechanics or the consideration of the after-comfort of operated patients.

DR. JOHN B. DEAVER: I have enjoyed Dr. Goldthwait's remarks, but must confess that I cannot endorse all he has said. It seems to me that with such a deformity of the scapula as mentioned by Dr. Goldthwait it will be found that Nature has provided for it in the shape of having made the ribs to conform. I believe strongly that Nature has prepared the human body in a most satisfactory manner.

My patients do not suffer from pain in the back to the degree that Dr. Roberts would have us think, and I believe it is because I do not give them morphine. If we could separate the morphine from the non-morphine-giving surgeons, we would solve the difficulty.

I have been much interested in the paper of Dr. Goldthwait, and again repeat, I am sorry I cannot more fully agree with the speaker of the evening.

DR. JAMES K. YOUNG: For three years past I have been making exploratory puncture in a large number of these cases of so-called specific arthritis, and in this manner determining the character of the infecting organism before operation. Diagnostic puncture and exploratory arthrectomy of the joints is of the greatest value in deciding the diagnosis.

In arthrectomy of the joints it will be found that in some the infecting organism has disappeared. In a case in which the knee-joint was recently opened there was no doubt that there had been gonococcic infection, but the infection had lost its virulence and the organism could not be identified from the joint.

DR. JOSEPH SAILER: I am particularly interested in Dr. Goldthwait's remarks relative to the value of binders and the various mechanical measures in these cases of neurasthenia with relaxation of the organs. I think it is obvious that in these cases, properly applied binders are of the greatest value. But if the patients are made to change their mode of life from a sedentary to active outdoor existence the results are often as good as are obtained from the application of binders. It has been taught usually that the benefit from the binders is the result of lifting up the internal organs, particularly the abdomen. *X*-ray studies do not very satisfactorily bear out this idea, because it can often be demonstrated that after the binder is applied and the patient relieved, the organs are in practically the same position as before. Dr. Goldthwait's explanation is suggestive. These binders should be applied tightly. They are passed around the back and as low as possible over the lower part of the abdomen by straps under the thighs. As a result they must tilt the upper part of the sacral region, relieving the strain from which those parts suffer.

Dr. Goldthwait's paper was in all its details extremely interesting, perhaps almost too plausible. His suggestion that the real trouble in splanchnoptosis is faulty posture rather than drags, kinks, or other conditions offers an interesting field for consideration.

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THE APPLICATION OF THE LUMIÈRE PROCESS OF COLOR PHOTOGRAPHY IN MEDICINE.[1]

By JAY F. SCHAMBERG, M.D.

It is my pleasure this evening to exhibit to you upon the screen photographs in color made with the Lumière autochrome plates, and to speak briefly of the uses of this ingenious photographic improvement in medical teaching. In the autochrome plate there is beneath the ordinary silver film a layer containing evenly distributed minute potato-starch granules colored with the three primary tints, red, blue, and green. The granules are so juxtaposed that the light passing through these minute colored screens produces the same impression upon the retina as if the screens were superimposed. In taking the photograph the exposure is made through the glass side of the plate instead of the film side, as in ordinary photography. In this manner the rays from a given object are permitted to pass through the color granules before they impinge upon the sensitized silver film. It is easily understood that the red granules will only permit the passage of red rays, the blue granules blue rays, and the green granules green rays. A red object, for example, such as a rose, would reflect rays which would pass through the red granules upon the corresponding portion of the plate, and, impinging upon the silver film would cause a deposit of silver which would render that portion of the plate opaque.

If the plate were now developed as a negative and viewed by transmitted light, each red granule over the affected portion of the plate would be covered by silver so as to obstruct the passage of

[1] Read April 1, 1908.

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light. When, however, such a plate is converted into a positive, the silver over the red granules is dissolved and light is permitted to pass through, whereas, all of the other granules are covered, and thus a red image is produced. As a matter of fact, the autochrome plates are always converted into a positive in the process of development.

As will be seen in the pictures upon the screen, not only are the primary colors of the spectrum reproduced, but also all intermediate shades developed from the union of the different rays. It will be noted that the colors portrayed approximate very closely the coloring of Nature. Inasmuch as positive plates are always made directly, these may, if a proper size be chosen, be employed as lantern slides. The use of the autochrome plates, therefore, will doubtless in the course of time be employed in the portrayal of all medical conditions which can be rendered by photographs in color. They will be found particularly useful in the teaching of diseases of the skin and the eruptive fevers. The disadvantages of this process at the present time are the expense of the plates, the duration of the exposures, which are much longer than those of ordinary photographic exposures, and the somewhat complicated technique of development. These difficulties, however, may be obviated in the course of time as improvements in the process are perfected.

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PHOTOMICROGRAPHS AND PORTRAITS OF DISEASES OF THE SKIN OBTAINED BY THE LUMIÈRE PROCESS OF COLOR PHOTOGRAPHY.[1]

By MILTON B. HARTZELL, M.D.

Dr. Hartzell exhibited a number of photographs and photomicrographs illustrating the clinical features and histopathology of a number of the commoner affections of the skin, made on the Lumière autochrome plates. He called attention to the fact that the color screen furnished by the makers of these plates is not suitable for photomicrography, since it shuts out the blue rays almost completely, probably owing to the fact that the balsam employed in mounting the sections, on account of its yellow color, also excludes a considerable amount of these rays.

DISCUSSION.

Dr. L. A. Duhring: I have listened with attention to the paper of Dr. Schamberg and that of Dr. Hartzell. The field is new, novel, and of much interest, and I believe that in due time, with improved technique and experience, it must develop into a valuable aid for the portrayal of disease in general. From what I have seen it is probably applicable to all diseased tissue, and not alone to the skin. The subjects before us, it seems to me, have been presented rather differently, one being the photography by this process from life, and the other from pathological specimens which have been stained, and from what we have seen the results are much more satisfactory when the picture is taken from life. As has been remarked by both the speakers, and especially by Dr.

[1] Read April 1, 1908.

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Hartzell in closing his paper, some of the pictures of cutaneous diseases leave but little to be desired, and I think even that little may be overcome in the future, when technique has been improved upon. We all recognize that there are many difficulties in the way of the technique, especially the correct exposure of the plates, and I know from my own experience in photographing disease that many more difficulties arise in hospital work than in a studio. I have taken many pictures in hospitals in former years, and whereas I had many failures under those conditions, when I worked in studios the results were much better. These photographs we have seen tonight have not been taken under the most favorable circumstances. I believe that both of the speakers, who have had a large experience in photography, would be able to get even better results in a studio properly lighted.

I would further say that the photomicrographs shown exhibit very little improvement over ordinary specimens colored by eosin, hematoxylin, and the like, and I fail to note where the new process applies strikingly to photomicrographs. The coloring is remarkable when one considers how it is done. I myself have not practised it, though I have been reading about it, and am glad to see this demonstration. The results are gratifying, and I believe that excellent work may be expected from this process in the future. The gentlemen are to be congratulated upon the success they have attained in the short time they have been working with this new process, and I am sure we are obliged to them for presenting this novel subject to the College this evening.

DR. SCHAMBERG: The Lumière color photographs appear to be far more satisfactory when viewed in the hand with the slide placed before the light of a white sky or before an illuminated ground glass than when projected upon the screen. Unless the positives are quite thin, the ordinary electric lantern does not give sufficient intensity of illumination to produce the most satisfactory results. One of the difficulties in securing a good photograph is the long exposure necessary. The average exposure of my plates in studio light has been forty-five seconds to one minute. As the warm months approach and the actinic intensity of the sun's light increases, the duration of the exposures may be lessened. It is obviously difficult to keep patients absolutely quiet for long exposures, and many plates are ruined through tremulousness of the subject.

In making photomicrographs I have omitted the use of the Lumière color screen, and find that the color is thereby better and more accurately reproduced. Dr. Hartzell observed that the yellow color of the Canada balsam in the slide acts practically as a color screen. I have developed photomicrographs at night time under an incandescent light reflector, and the results have been equal to those obtained by development in daylight. It is not possible at the present time to make color prints upon paper. It will be recognized that the positive is viewed by transmitted light, whereas a print upon paper must be seen by reflected light. Thus a considerable obstacle exists to the development

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of printing in color. However, the difficulty does not seem to be entirely insurmountable, and we may hope that the ingenious inventors of the Lumière process may be able to achieve the long-wished-for consummation of photographic prints in natural colors. The Lumière plates must be developed in absolute darkness, as they are panchromatic and sensitive to the red light of the photographic dark room. The cost of the plates is rather high, but there is reason to believe that their expense in the course of time will be lessened.

I desire to congratulate Dr. Hartzell on the excellent photographs that he has been able to secure by this method.

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MYOSITIS OSSIFICANS PROGRESSIVA.[1]

By WARREN WALKER, M.D.

Millie R., aged seven years, born in Philadelphia. Italian parents. Has three sisters and four brothers, all living and well. Father and mother living and well. No history of tuberculosis, syphilis, or rheumatism in family, with the exception of an uncle who has rheumatism.

She has never had any diseases of childhood. At birth child was perfectly formed, except the great toes were shorter than the second toes and bent outward. No special attention was paid to this. The child has played about as every other youngster of her age would, until July, 1907, when she developed a stiff neck which was treated with ordinary remedies without effect. In August a swelling was noticed in the right pectoral muscle which was hard, and the skin of a dark-red hue and painful to the touch. Between August, 1907, and February, 1908, when I first saw her at the Children's Hospital dispensary, she had had several attacks of pain, swelling, and redness in different locations on the back and upper extremities, each followed by exostoses or hardening of the muscles.

Examination February, 1908. *Head:* Normal with no involvement of the temporal or masseter muscles.

Neck: The first thing noticed is the marked prominence of the occipital tendon of the right trapezius muscle. It is very hard and stands out like a cord. The scaleni and sternocleidomastoids are also involved, but not to such a degree as the trapezius. The head is fixed, with chin pointing down within two fingers' breadth of the sternum. No rotation of the head is possible.

Back of Trunk: Upon examination of the back one is struck by the exostoses (Fig. 1). The most prominent is at the lower angle of the left scapula. It is about one inch in height and one-half inch in width. A smaller one is not quite so prominent, and is situated on the ninth rib below the one described above. The left scapula can hardly be outlined owing to the marked induration of all the muscles about it. The condition of the right scapula is about the same; there are two exostoses below it. The larger being round and situated about one

[1] Read May 6, 1908.

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inch from the spine, the smaller above it and a little to the outer side. There are several exostoses on the eleventh and twelfth ribs on left side, and several on the left side of the lumbar spine in the substance of the erector spinæ muscle (Fig. 2). The spinal column is fixed and the child has great difficulty in picking up an object from the floor.

Upper Extremities: We are struck by the lack of motion and the position in which the child holds her arms. They are held away from the sides in partial abduction, this being more marked in the left than right. Neither elbow can be brought in contact with the sides. The left humerus is fixed, the only motion being that which the movement of the scapula allows extension, abduction and adduction being impossible (Fig. 2). On the shaft of the left humerus there is a pigeon-egg-sized bony mass at the inner border of the belly of the biceps muscle. The elbow can be flexed to a right angle, and can be fully extended. Motion of the right arm is limited; it cannot be fully abducted —extension to about 45 degrees. Flexion of elbow is possible to about 45 degrees. The loss of motion in the arms is due to bony masses in the axillary muscles. Palpation of the axilla shows bony masses in the anterior and posterior folds of axilla. Those in the posterior fold being in the substance of the latissimus dorsi muscle. The *x*-ray shows that the bony masses in the left axilla apparently arise from the fourth rib and extend upward and join the humerus in its upper 1 to 3, and in this way prevents motion in the left arm. Those in the right latissimus dorsi muscle have not united with the humerus, thus accounting for the freer motion on that side (Figs. 2 and 3). The deltoids and biceps are not involved.

Hands: The fingers are all short, the little fingers being markedly so. No ankylosis or exostoses.

Front of Trunk: The muscles of the right side are more indurated than those of the left. There are no bony masses found in their substance or on the ribs anteriorly. There is entire absence of respiratory motion in the chest, the breathing being entirely diaphragmatic. The abdominal muscles are not involved.

Lower Extremities: Walking not interfered with. No lesions of the muscles found. No exostoses found. The left thigh three-fourths of an inch larger than the right. Both great toes show microdactylia, being fully one-half inch shorter than the second toes. A condition of hallux valgus is present. The interphalangeal joint is ankylosed and the first metatarsal bone is short and stubby (see *x*-ray and photo., Figs. 4 and 5).

This condition, which is a very rare one, was first described by Freke in the *Philosophical Transactions* in 1740. It was described more thoroughly and given its present name by Munchmeyer in 1869. Since that time nothing has been added to his description

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FIG. 1.—Showing exostoses and full amount of abduction.

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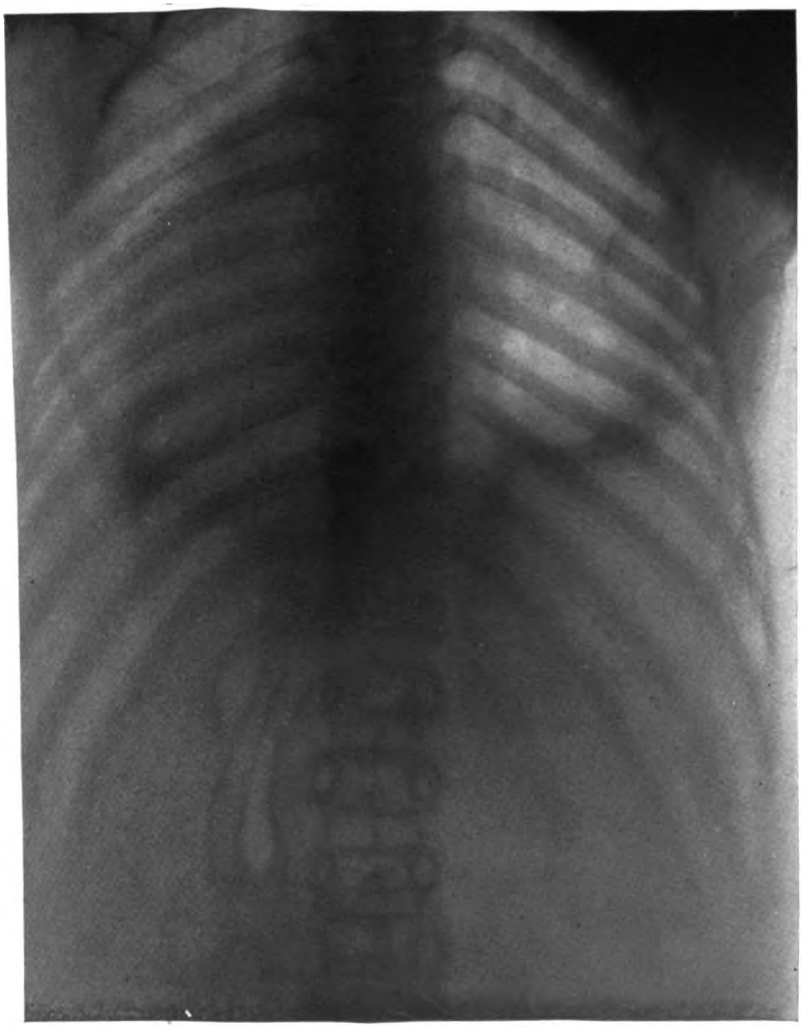

FIG. 2.—*X*-ray showing bony deposits in latissimus dorsi muscles and in erector spinæ of left side.

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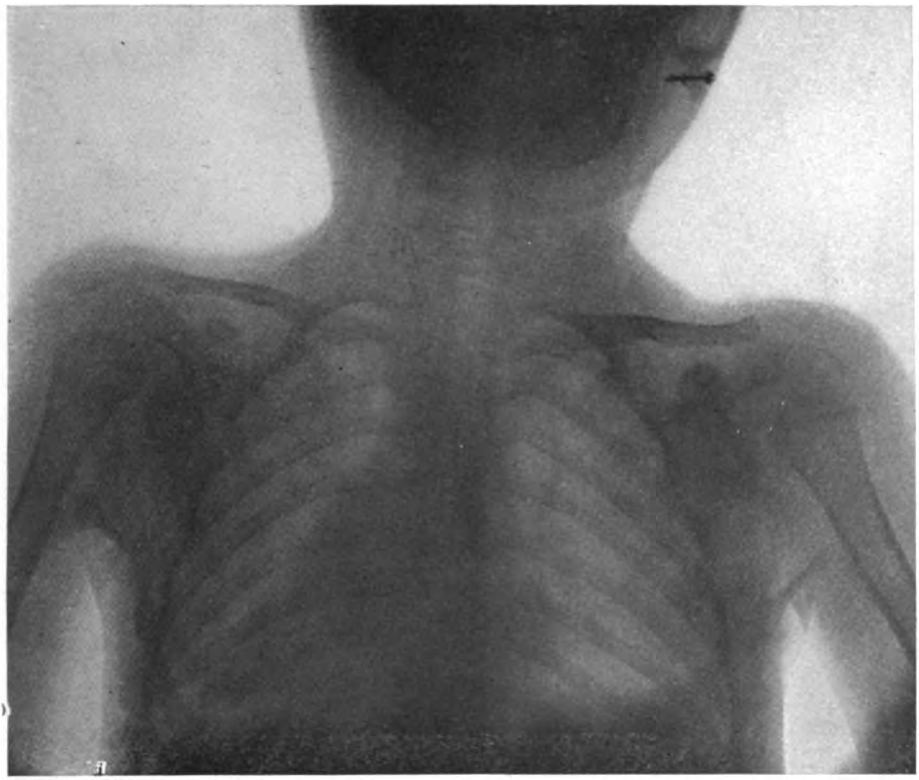

FIG. 3.—*X*-ray bony deposits in substance of latissimus dorsi muscles.

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FIG. 4.—Showing microdactylia.

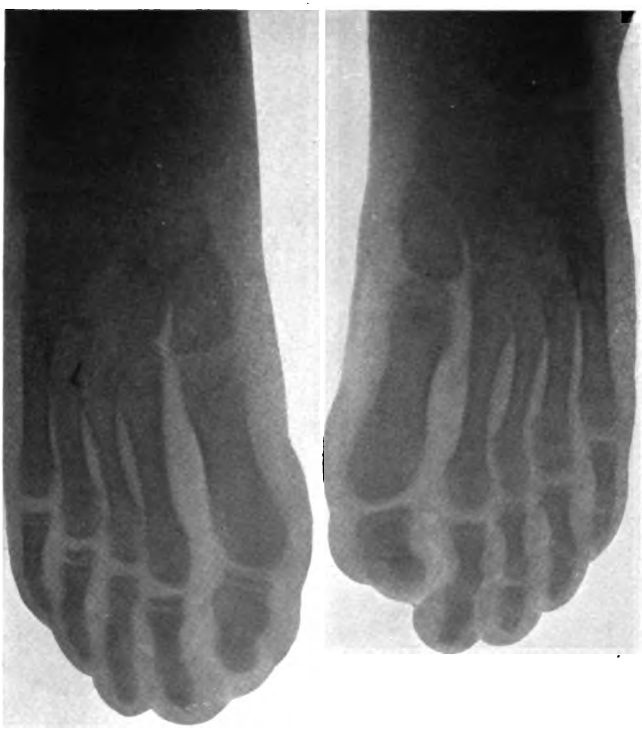

FIG. 5.—*X*-ray of feet of child suffering from myositis ossificans showing microdactylia.

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except the condition of microdactylia, which was pointed out by Helferich in 1879. This consists of a shortening of the thumb and great toes. This is due to a shortening of the metatarsal bone. Ankylosis between the two phalanges is very common, and before the days of *x*-rays the condition resulting from ankylosis was described as absence of one phalanx. The great toes point outward and in some cases there is a partial dislocation at the metatarso-phalangeal articulation. This condition appears to be a congenital deficiency and bears out the theory that the disease is a congenital one depending on some congenital aberration of growth.

There are two forms of the disease:

1. Local or stationary form.
2. Progressive.

The progressive distinguished from the local because it develops in youth—advances periodically and attacks many muscle groups.

The disease has been reported in nearly every country, but seems to be much more common in America, England, and Germany—fully 80 per cent. of the cases reported being from these three countries; only six have been reported from France.

Rolleston claims it to be five times more frequent in boys than girls. Nové-Josserand says 3 cases in girls to 4 in boys. It usually begins in the first ten years of life and progresses slowly with intervals of quiet, death occurring in ten or twelve years—either from some intercurrent disease, especially bronchopneumonia, or from inanition due to involvement of the masseter muscles.

The muscles of the hands, the feet, face, inner ear, diaphragm sphincter, heart, tongue, and genital organs are not attacked.

In a very complete paper by De Witt in 1900, 78 cases of the progressive type were reported. Since that time I have found 13 cases reported as follows: Comby and Davel, Ferranton, Graham, Menard and Tallaye, Micheli, Michelsohn, Nové-Josserand and Horand, Rager, Rivalta, Rolleston, Salvetti, Soncini, and Wilkinson. This case makes 14. On analyzing these I find that the disease occurred five times in boys to six in girls. Three cases I did not have access to. The average age was about six years, the youngest being two and one-half years and the oldest

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eighteen years. This last being the age at time of examination, age at beginning of disease not being given. Ten of the 11 cases showed microdactylia to be present.

There are three principal clinical facts which characterize myositis ossificans progressiva according to Menard and Tillaye:

1. Localized bony masses.
2. Evolution by congestive eruptions.
3. Existence of troubles of development.

Localized Bony Masses. The localization is almost always the same, the trunk, the upper extremities, and the neck. In only one instance has a bony mass been discovered in the lower extremity.

Numerous muscles have been ossified in the upper extremity, but only in the arm and forearm; the hand has never been involved. The deltoid, biceps, brachialis anticus, and the muscles inserted in the bicipital groove of the humerus and pectorals seem to be most involved. They are usually only partially ossified and chiefly at their upper insertion.

In the neck the muscles chiefly involved are the sternocleidomastoids, the scaleni, hyoids, and upper fibers of trapezius. The ossifications of the muscles of the neck has caused this disease to be mistaken for torticollis and suboccipital Pott's disease.

Of the face muscles the masseters are alone affected. When this occurs the prognosis is very grave, as alimentation is interfered with.

In the great majority of cases at least one muscle of the back is involved, those usually involved being latissimus dorsi, trapezius, rhomboids, and serratus magnus—supra- and infraspinatus.

The evolution of myositis ossificans progressiva by congestive eruptions constitutes one of its principal characteristics. A child in perfect health develops in a localized area a cyanosed condition; the tissues in the area are of doughy consistency and painful. This inflammatory condition is absolutely local, there being no elevation of the general temperature. The digestive, pulmonary, circulatory, and nervous system are normal. These eruptions occur several times in the same location, the length of time between attacks varying, each ensuing attack adding more to the indura-

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tion of the muscle or muscles attacked. A muscle never becomes ossified without an attack of this sort.

The third clinical fact characterizing myositis ossificans progressiva consists of developmental troubles. Microdactylia of the hands and feet, ankylosis of the interphalangeal articulation of the great toe, absence of the first phalanx of the great toe, hallux valgus and malformations of the genital apparatus. These malformations have led certain authors to class myositis ossificans progressiva among the diseases of congenital origin; others have thought the disease might have an inflammatory, neoplastic, or traumatic origin. It could hardly be of an inflammatory origin, as the skin of the affected area is never red but more of a violent hue, and there is no fever, and the absence of those cardinal symptoms, redness and heat, eliminates the inflammatory origin.

Concerning the neoplastic origin myositis ossificans progressiva has been compared with multiple exostoses originating without pain or redness. In multiple exostoses muscular troubles are not encountered, nor are any arrests of development which are always present in myositis ossificans progressiva. In myositis ossificans progressiva the exostoses are always secondary to the ossifications of the muscles, and, moreover, the exostoses of myositis ossificans progressiva are sometimes absorbed; this fact would be difficult to explain if it were a neoplasm.

As to the traumatic origin, the most that can be said for this is that it may be an adjunct, as it is wanting in a great majority of cases. Congenital syphilis has been thought by some to be a cause, but specific treatment has been given without any benefit.

The treatment of these cases has been very unsatisfactory, no remedy having been discovered which affords any relief, although a great many have been tried.

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DISCUSSION.

DR. J. K. MITCHELL: The patient is an interesting one. As to the question of etiology which Dr. Walker has discussed, I have had two cases for comparison, not of the same disease, but of allied conditions. One I showed before the College, of multiple exostoses, which so far as we could discover had no determining cause, although the family said the first growth had appeared during or after the course of typhoid fever. A number of exostoses in different parts of the bones had developed. There were none in the muscles. I have recently seen a patient with progressive fibrous myositis. I mention this because, when we examined muscle fibers removed from one of the posterior group of thigh muscles, we found a distinct degenerative fibrous change going on. There was no apparent ossifying change. The muscles were capable of contraction which would move the leg through an arc of about 8 or 10 degrees, no more, contracting the knee on the thigh to that extent; then they stopped and could not be contracted further, and at this point they were of a density almost comparable to a bony growth. When relaxed they did not seem of quite so great a density, but still showed great hardening and thickening, so marked that Dr. Spiller, who saw the case, expressed the opinion that this was the preliminary stage of what would in the future become an ossifying process, though I cannot see in the pathological specimen any reason for that belief. The patient went home considerably improved in his condition, and three days after his return got pneumonia and died. Unfortunately, I was not informed until two days after the funeral, or I should have endeavored to secure a more satisfactory specimen to complete the examination.

DR. W. M. L. COPLIN: I wish to allude to a few points concerning these cases, and especially three interesting ones which have come to my knowledge. In one of these the masses had been excised, examined microscopically, and pronounced giant-cell sarcoma. In another case a limb amputation, I think, was considered; at a second operation practically all the involved tissue in one of the adductors of the thigh was excised. I do not remember exactly what part was removed; I did not see the case, only a part of the specimen. The resem-

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blance in some of the sections to giant-cell sarcoma was so great that it was easy to see how one could be misled. I also know of an instance where the scapular group of muscles was affected; because of the richness of the mass in giant cells and the rather cellular character of the growth as studied in frozen section at operation, the diagnosis of giant-cell sarcoma was made; the calcifying masses—the calcified nodules scattered through it—would not be at all in discord with the view that it was sarcomatous. In two of these cases I had an opportunity to examine there was striking basophilic reaction in the protoplasm of the giant cells, suggesting very stongly the reaction of myeloblasts of the bone marrow. This of course indicates that the osteophytic growth may, in some way or other, be related to bone extension, and it is well known that in the traumatic form the relation to the periosteum is very close; especially is this true in the muscles of the shoulders and thigh. The relation to trauma is very well established and also well known, and is most familiarly illustrated in cavalrymen and riding masters. In all cases there appears to be some relation to periosteal growth; in this case the development of exostoses suggests that there is something of an osteogenetic process seen in the peripheral portion of bone, and, of course, of periosteal origin. The important point I wish to bring out, however, is the fact that excised portions of the involved tissues have been mistaken for sarcoma, and that such an error in histological diagnosis is most readily made.

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THE TREATMENT OF IRREDUCIBLE CONGENITAL LUXATIONS OF THE HIP BY OPERATIVE MEANS.[1]

By GWILYM G. DAVIS, M.D.

THE treatment of congenital luxations of the hip by the bloodless method of reduction has so occupied the attention of the profession for many years past that it may be well to recall what can be done for the relief of those cases which have passed beyond the period when it is possible to replace them by non-operative procedures. Many such patients pass their earlier years with comparatively little pain, and, notwithstanding their deformity and more or less disability, do not apply for treatment until pain and difficulty of locomotion demand relief. The history of the cases are briefly as follows:

CASE I.—Jessie V., aged fifteen years, was admitted to the Orthopedic Hospital October, 1904, with double congenital luxation of the hips. While both hips were affected, one only, the left one, was causing complaint. The right one seemed firmly applied to the ilium and did not appear to move in walking. The left one, however, shifted its position markedly. It was quite loose, and the left limb was 3 cm. shorter than the right. There was marked waddling gait and pain, which caused her to apply for relief. She was put to bed and weights amounting to fifteen pounds were applied for about six weeks. In December, 1904, she was etherized and an unsuccessful attempt was made at reduction by the ordinary circumduction method. Hyperextension over König's padded block was also tried, without result. A heavy, wide, close-fitting belt of webbing with a pad and perineal straps was then applied, which afforded some relief, and she left the hospital.

In February, 1906, two years later, she was readmitted, with her condition much aggravated. Pain was almost constant, walking for any distance was

[1] Read May 6, 1908

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impossible, and she was unable to earn her living. Her charitable friends were then looking around for some place where she could be taken care of. She was again put on extension, and again an unsuccessful attempt was made at reduction. In April, 1906, now over two years ago, she was operated on. An anterior incision was made, a new socket was excavated from the ilium above the old acetabulum, and the head inserted therein and the limb put up in plaster of Paris in an abducted position. She was kept abed for eight weeks and then allowed to get up with a cast on. This was removed one month later, and in another month she was walking well with the aid of a cane. She was then discharged. Since that time she has improved, and, while still having a somewhat peculiar gait, can walk long distances without pain. The hip is movable in all directions, but does not slide up and down; it has evidently retained its position in its new acetabulum. She was formerly unable to take exercise, and grew fat and very heavy; now she is active, working and earning her living, and has lost the excessive fat and weight which previously encumbered her. The transition from her previous helpless condition to her present active and useful one has been very marked.

CASE II.—Minnie H., aged sixteen years, was under treatment in 1892 by another surgeon for some deformity of the feet, the exact character of which is not known. It was stated by the nurse that when an infant the child had paralysis of both lower extremities. Tenotomy of both tendo Achilles was done, and later braces were applied and worn continuously thereafter.

In 1897 she came under my care. At that time there was marked paralysis of both lower extremities, together with double congenital luxations of the hip. Locomotion was almost impossible. One foot was in marked paralytic valgus, while the other was more of a paralytic equinovarus. The left hip was luxated anteriorly, while the right was luxated posteriorly. An arthrodesis was done on the right foot, which improved its position somewhat. The right hip was then operated on by an incision over the trochanter; the ligamentum teres was removed and the adductors and tensor fasciæ femoris divided subcutaneously. The head was brought down to the acetabulum, but as it would not stay in place a new acetabulum was excavated. The child was markedly strumous, and long-continued suppuration followed this operation. It did not involve the deep parts around the bones, but rather the superficial parts, so that, while very extensive scarring resulted, the hip region was not at all stiffened and movements were not restricted. After two months, however, she was gotten up on a brace, and she was able to get around some, but dragged especially her left leg. Nothing was done to the left hip, which remained fairly well in its displaced position, with the head just below the anterior superior spine and the trochanter posterior; evidently there was a twisting of the neck and shaft.

She again applied for treatment in 1906, nine years after the previous oper-

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ation. On examination the right hip was found to have again become luxated on the dorsum of the ilium. The left hip was apparently somewhat more posterior. The right hip slid up and down from one-half to one inch. In April, 1907 (thirteen months ago), the right hip was again operated on, a new acetabulum was dug out and the head inserted, and the limb put up in the abducted position in plaster of Paris. Healing was uneventful. To correct the dropping down and in of the anterior part of the right foot the tibialis posticus, extensor proprius hallucis, and peroneus brevis were loosened and attached to the extensor longus digitorum. The distal end of the extensor proprius hallucis was attached to the tendon of the adjoining toe.

On the left foot the peroneus brevis tendon was attached to the tibialis anticus, as were also the extensor tendons of the fourth and fifth toes. The distal ends of these tendons were sutured to the tendons of the second and third toes.

After about twelve weeks the patient got around. Stout shoes were ordered to hold the feet firmly, and a corset to support the trunk.

At present, one year after operating, she walks without cane or crutch. She has a marked paralytic eversion of the left foot, with evident twist in the bones. An apparatus might be constructed to hold this foot inward, but it would have to extend from the pelvis to the foot, and this she declines to wear. The hip is firm in place and movable functionally, and gives her no trouble.

CASE III.—Miss C., aged twenty years, had a congenital luxation of the left hip. She limped, but not excessively. About five years previously, while walking, the joint seemed to catch, and since then it ached, and the pain became so constant as to induce her to seek relief. This case was operated on in conjunction with my colleague, Dr. Taylor. An anterior incision was made, as in the other cases, and a place was cleared in a depression in front of the former location and above the acetabulum, but no bone was removed. The limb was put up in plaster in abduction, at present over a year after the operation, and she walks excellently. There is no slipping of the head of the femur, and with a half inch added to the heel she is comfortable, unless she unduly exerts herself, when she experiences a feeling of weakness.

These cases represent some of the worst in the domain of surgery. When a person is young, say under ten years or so, the dislocated hip can usually be replaced without a cutting procedure, but after that age attempts at reduction are apt to fail, and other means have to be resorted to. Some surgeons do not regard an unreduced congenital luxation as being very serious. My experience has been different. In almost every case a greater or less amount of disability occurs in later life in addition to the awkward gait and limited use

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of the affected joint inseparable from the affection. Only recently have I seen a woman, aged fifty-five years, again disabled by pains in the affected hip.

A disabled hip is an unreliable hip that is certain to give rise to serious trouble in some portion of the patient's life. These congenital luxations can usually be reduced by the bloodless method up to about the age of ten years. Later it is in most of the cases impossible to restore the head to its socket, and one is therefore forced to seek a new position for the head of the femur to rest in.

The treatment of congenital hip luxations has developed on two separate lines—the non-operative and the operative. It began about 1826, when Dupuytren brought forward his pelvic band. Corsets, jackets, and braces followed. The next advance was about in 1847, when Humbert, and especially Pravaz, of Lyons, advocated traction to bring the hip down and then pushing it over into place. This method was followed in a more or less modified form by Buckminster Brown, Bradford, Mikulicz, Max Schede, and others. While these traction methods were being developed, other surgeons were doing operations for the same conditions.

Guerin was one of the first to cut the muscles subcutaneously; Brodhurst and others followed. Resection of the displaced head was given quite a thorough trial, until, finally, on January 29, 1888, Alfonzo Poggi, of Bologna, replaced the head in a new acetabulum scooped out of the side of the ilium. Two years afterward the late Prof. Hoffa brought out his well-known method of detaching the muscles about the great trochanter and making a new acetabulum. In 1892 Lorenz modified Hoffa's procedure by making an incision from the anterior superior iliac spine downward and outward along the posterior edge of the tensor fascia femoris muscle. In 1895 Hoffa, having been convinced by Trendelenburg that loosening of the muscles attached to the greater trochanter was unnecessary, made an incision along the anterior edge of the greater trochanter from its tip down. There have been practically no further developments of the open operative method since that date. While the bloody method was occupying the attention of the surgical world, Agostino Paci, of Pisa, was publishing his work by the bloodless

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method from 1888 to 1893. In 1894, at the International Congress in Rome, Lorenz and Hoffa came prepared to fight for their respective cutting operations, but Paci by this time had systematized his work. He gave his results on 23 cases of congenital luxations, besides 5 of pathological and old traumatic ones; also those of ten other surgeons who had used his method. He submitted a specimen showing a perfect antemortem reposition, and wound up by performing his bloodless method on a patient before the assembled surgeons. The effect might almost be described as ludicrous. No more was heard of cutting operations. Lorenz, in the following year, modified Paci's procedure, and travelled far and wide exploiting it.

The majority of congenital luxations of the hip which one now sees can be replaced by the more or less modified bloodless method of Paci, but occasionally a case is seen which is impossible to so reduce.

A person who has only one hip luxated, as in the third case, not infrequently has such an amount of disability as to demand relief. Should such a case not receive attention before, say, the age of ten or twelve years, it varies considerably; then the only other alternative is an open operation, or they must bear their ills as best they can. If the case happens to be a luxation of both hips, one will be apt to be much worse than the other. This was the condition in Case I. The right hip, though luxated, was still fairly fixed in its displaced position; the left, however, was so loose and caused such pain as to absolutely disable the patient and prevent her from earning her living. In the second case there was not only a luxation of both hips, but also the results of an infantile paralysis involving both lower extremities, as well as the muscles running from them to the trunk. Of course, all the muscles were not paralyzed; some escaped, and by producing an artificial ankylosis in one foot (arthrodesis) and transplanting tendons in both feet, the legs were made to acquire a certain degree of stability. The effect of the paralysis of the trunk muscles was partly counteracted by means of a firm corset. The left hip at first was luxated upward and anteriorly, the head being just below the anterior superior spine; later, it

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has assumed a somewhat more posterior position. It was not, however, loose like the hip on the right side. The deplorable condition of this child, with both hips luxated, together with the extensive paralysis, can be readily appreciated.

In 1897 I had operated on the left hip of this patient by Hoffa's original operation, but several years later it was found to have relapsed. In 1899 I showed before the Philadelphia Pediatric Society a child a little over five years of age, in which one hip had been replaced by the bloodless, and the other by the open, operation, with success in each case.

In each of the three cases reported to-night the object was to fix the head of the femur in a new acetabulum, so that in walking the weight of the body would be borne by the bones and not by the soft parts, and this result was accomplished. In operating for this affection, the incision I prefer is an anterior one, straight down from the anterior superior spine. Hoffa's incision was along the anterior edge of the greater trochanter, while that of Lorenz ran downward and backward from the anterior spine. The reason the extreme anterior incision is preferred is because of the better access which is obtained. The lateral incisions are difficult to separate in order to work properly. In the anterior incision, if more room is necessary, we can do as advised by Codivilla—separate the structures from the crest of the ilium from the spine back as far as desired. On the completion of the operation they are to be reattached by means of sutures of chromic catgut. To deepen the acetabulum, I prefer the gouge and burr devised several years ago.[1] The gouge (Fig. 1) has a round edge and is sharply bevelled on its outer side. It cuts quickly and efficiently, and with it the chips are easily removed.

The burr (Fig. 2) is one inch in diameter and has ten cutting teeth around one-half of its circumference only. The cutting side is used to smoothe and deepen the new acetabulum at any desired spot, while the smooth side presents toward the femoral head the cartilaginous covering of which thereby remains uninjured. In order to facilitate lowering the head, a lever (Fig. 3) was devised, consist-

[1] Trans. Amer. Orthop. Assoc., 1899, p. 331.

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ing of a rod bent at a right angle, having a square handle at one end and a small curved plate at the other. By loosening the screw holding the plate on, the latter can be removed and reversed for

FIG. 1.—Gouge.

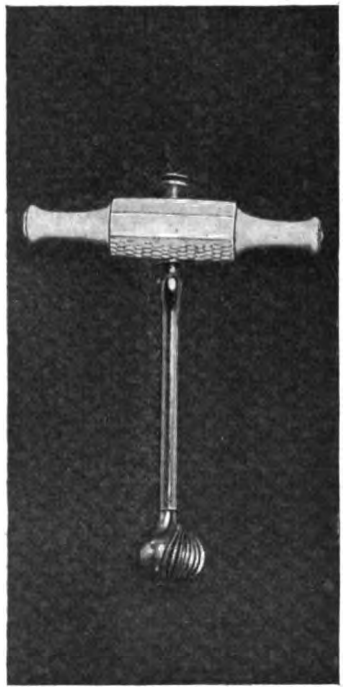

FIG. 2.—Burr.

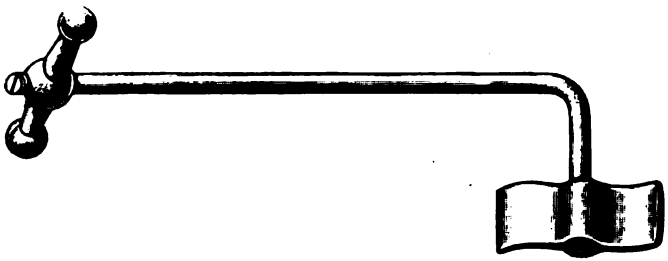

FIG. 3.—The lever for lowering the head of the femur.

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use on the opposite hip. The smaller plate is 4.5 cm. (one and three-quarters inches) long, and is intended for children; the longer is 6 cm. (two and three-eighths inches) long, and is intended for adults. A firm, square handle is essential to manipulate the instrument properly.

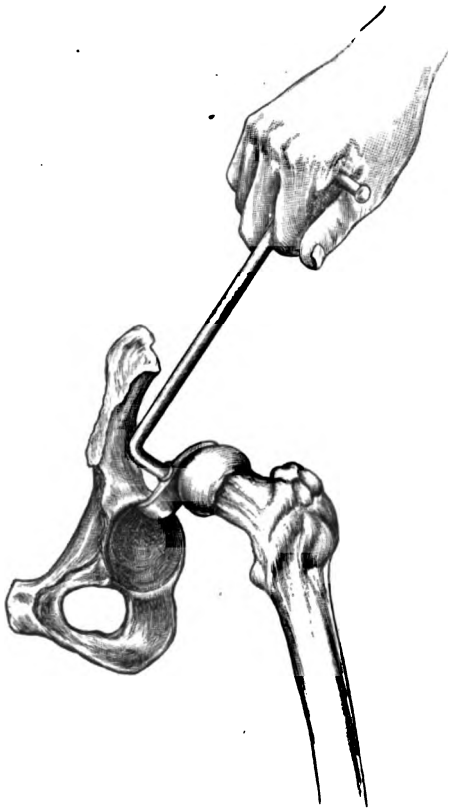

FIG. 4.—Method of replacing the head of the femur.

In using it, one end of the plate is hooked under the edge of the acetabulum, while the other end rests on top of the femoral head. By depressing the lever the head slides along the shallow groove of

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the plate into the acetabulum (Fig. 4). A. Codivilla, of Bologna, used a straight lever for this purpose.[1]

In order to dilate and stretch the capsule and adjacent fibrous structures, the uterine dilator of Pryor (Fig. 4) has been found of service. It is very strongly constructed.

The incision used passes almost directly downward from the anterior superior spine of the ilium; this is between the anterior or inner edge of the tensor fasciæ femoris and sartorius muscles. We prefer this to the incision of Lorenz, which followed the posterior edge of the tensor fasciæ femoris and to that of Hoffa, who made

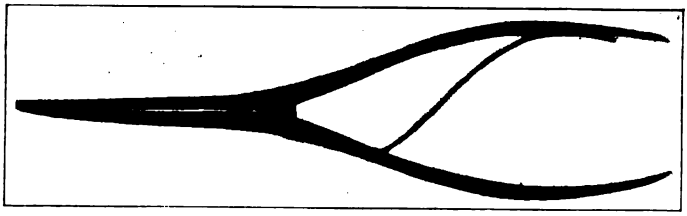

FIG. 5.—Uterine dilator of Pryor.

one along the anterior edge of the greater trochanter. One should avoid as far as possible injuring the branches of the deep circumflex artery and nerve as they cross the femur. Drainage may be used for twenty-four hours and the limb placed in a position of marked abduction in plaster of Paris.

After eight weeks the plaster is to be removed, the limb brought down somewhat, and the patient allowed to go about on crutches.

Over two years have elapsed since the operation in the first case and over a year in each of the other two, so that there is reason for hoping that the present results will be permanent. In order to avoid ankylosis, it is in the highest degree desirable not to wound the cartilage on the head of the femur.

[1] Archives di ortopedia, 1902.

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DISCUSSION.

DR. E. B. HODGE: I think it might be interesting for the members to know that Dr. Ridlon, of Chicago, saw the second case Dr. Davis presented, and said he could not do anything for her. He advised making no attempt to relieve her. Dr. Davis was too modest to allude to this, but I think it emphasizes the success of Dr. Davis' treatment.

DR. A. P. C. ASHHURST: Too many surgeons are still imbued with the idea that it is sufficient to let these conditions alone, and that the patients will get along very well without operation. Dr. Davis' cases illustrate the falsity of this reasoning. A number of years ago we had a patient come to the Orthopedic Hospital, in Dr. Harte's service, and she was directed to wear a high shoe, a form of treatment which was considered at that time to be sufficient for such cases. The patient wore the high shoe for some years and then she came back; she was not satisfied. So we had to set to work and reduce her dislocation, when she had long passed the age at which reduction is easily accomplished. The patients shown here to-night by Dr. Davis certainly present a vast improvement over their previous condition. The second patient, the Italian girl, came first to the Episcopal Hospital, where I saw her, and referred her to Dr. Davis, and I can bear very earnest testimony to her lamentable condition at that time. She had a "sea-going" gait, wobbling from side to side, and with difficulty able to walk a half square. The other patient shown by Dr. Davis, as Dr. Hodge has just said, was pronounced hopeless by one of the most eminent orthopedic surgeons in this country.

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A CASE OF IRREDUCIBLE CONGENITAL LUXATION OF THE HIP TREATED BY OPEN OPERATION.[1]

By GWILYM G. DAVIS, M.D.

THE case presented is one of three which were reported to the College at a late spring meeting. The other two cases were shown at that time, and this patient is the third. I had the pleasure of being associated in this case with my colleague Dr. Wm. J. Taylor.

The patient is a young woman, aged twenty-odd years. She applied for relief from pain. This pain was excessive and was not only present while walking, but at all times, not disappearing even on remaining off her feet for considerable periods of time. She was practically disabled. The operation which was performed consisted in making an incision directly downward from the anterior superior spine of the ilium. The tensor fasciæ femoris and glutei medius and minimus muscles were pulled outward, and the sartorius, iliacus, and rectus femoris inward. This exposed the joint capsule, which was then detached from the bone. As it was impossible to bring the head down to the site of the remains of the acetabulum, the ilium just above was cleared off and the head of the femur placed in a depression of the bone at that point. A new acetabulum was not gouged out of the bone. The wound was closed and the limb put up in plaster in a position of extreme abduction. Healing was uneventful, and she was finally discharged from the hospital. It is now a year and nine months since the operation was performed, and sufficient time has elapsed to enable us to form a fair estimate of the probable result.

As regards walking, it is evident that she possesses a barely perceptible limp. This is rendered possible by the use of a slight increase in the height of the heel, a rubber heel being added to her ordinary shoe. This compensates for the shortening on the affected side. She uses no crutch, cane, or other artificial support, either in the house or out in the street. Her pain has absolutely disappeared, and there is no longer the "catching" sensation she previously

[1] Read November 4, 1908.

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experienced when walking. The joint is freely movable. She states, however, that she is not so comfortable walking on the streets as in the house. This she attributes to her knee, which she says is placed in extreme extension with a little difficulty. A possible explanation of this is that the tension of the hamstring tendons—due to bringing down and forward the head of the femur and placing it in its new position—has not yet entirely disappeared. This will probably occur as the limb is used. She has pursued her occupation uninterruptedly for a long time past.

This is only one of many cases which have come under my observation of serious disability occurring in those cases of congenital luxation of the hip which have been left to progress untreated. It shows the necessity of doing something to aid this class of sufferers. It is also offered as evidence that the pessimistic views expressed by many writers in regard to operative measures in this class of cases is not justified. In my experience the results have been very gratifying. I regard the operation and after-treatment as being difficult and tedious, but well worth one's while.

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THE OPERATIVE TREATMENT OF PAPILLO-EDEMA (CHOKED DISK), WITH SPECIAL REFERENCE TO DECOMPRESSING TREPHINING.[1]

By G. E. de SCHWEINITZ, M.D.,
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AND
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In recent years the treatment by operation of those alterations which take place at the intra-ocular end of the optic nerve as the result of increased intracranial tension or pressure has attracted widespread interest. Concerning this matter, Sir Victor Horsley writes: "That the release of intracranial tension arrests and cures optic neuritis was first published by myself about twenty years ago, and this has been confirmed by very many surgeons and physicians during that period."[2] Our own attention was first called to this subject by Dr. William G. Spiller at a meeting of the Neurological Society in 1901,[3] and in America we are much indebted for advances along these lines to Drs. W. W. Keen, M. Allen Starr, C. K. Mills, W. G. Spiller, F. X. Dercum, C. H. Frazier, W. J. Taylor, E. Martin, Harvey Cushing, and a number of other surgeons and neurologists whose work has been reviewed by Spiller in the *University of Pennsylvania Medical Bulletin*, September, 1906, and in the *Transactions of the Ophthalmic Section of the American Medical Associa-*

[1] Read November 4, 1908.

[2] The Ophthalmoscope, 1908, vi, 658.

[3] Journal of Nervous and Mental Disease, May, 1901. Writing in 1896 (New York Medical Record, February 1, 1896), M. Allen Starr called attention to the relief of optic neuritis and other brain tumor symptoms by removal of a portion of the skull, as advocated by Horsley, and describes an operation of this character in a patient with tumor of the corpora quadrigemina. Headache was relieved, but there was no effect on the optic neuritis.

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tion, June, 1908. Important recent foreign communications are those by Leslie Paton,[1] von Krüdener,[2] and E. von Hippel,[3] but as this paper is intended to record simply a personal experience based upon a series of cases which are hereafter quoted, we have not thought it necessary to analyze any of the previous writings upon this subject, but have contented ourselves with the brief references that have already been made.[4]

First, a word as to the pathogenesis of the intra-ocular changes which occur under the influence of increased intracranial pressure. Numerous theories have been propounded, and a review of them at the present time would be unprofitable, because much modern investigation, so greatly helped by the results of cerebral surgery indicates, as Herbert Parsons[5] has said, that "all those who have had opportunities of watching the extraordinary effect of the relief of intracranial pressure upon a choked disk must agree that no theory which leaves this element out of account requires any further consideration," or, as Sir Victor Horsley[6] maintains, "I would point out, first, that optic neuritis is produced by a combination of factors of which the only one we know to be certainly present is a rise of intracranial tension or pressure." It would, therefore, seem that under most circumstances the views of Schmidt-Rimpler and Manz are correct, namely, that the marked edema of the nerve head, which is so conspicuous a symptom in the condition under consideration, is primarily due to a distention of the sheath of the optic nerve, caused by the increased subarachnoid fluid being forced into this situation under the influence of elevated intracranial pressure, and that, as Bordley and Cushing

[1] Transactions of the Ophthalmological Society of the United Kingdom, 1905, xxv, 129; and ibid., 1908, vol. xxvii, p. 112.

[2] Archiv f. Ophth., 1907, vol. lxv.

[3] Bericht über die XXXV Versammlung der Deutschen Ophth. Gesellschaft in Heidelberg, August, 1908.

[4] We are indebted to Ward Holden for the following extremely interesting quotation: "When the sight fails in a person in good health, one must cut down upon the bone at the vertex, draw aside the skin, trephine the skull, and permit the escape of the exuded liquid. In this way the sight of the blind is restored." (Hippocratic Writings, C. 8.) Hirschberg gives the original Greek in Graefe und Saemisch, Handbuch der Gesamten Augenheilkunde, Teil II, Band xii, Kap. xxiii, p. 94.

[5] The Pathology of the Eye, 1908, iv, 1363.

[6] Ibid., p. 658.

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have written, "since there is an almost uniform subsidence of what we consider to be choked disk (barring certain cases complicated by destructive hydrocephalus), after depressing operations, whether conducted for the pressure of a tumor, cerebral edema of one sort or another, infections or intracranial hemorrhages, a mechanical rather than a toxic process must play the chief rôle in the causation of this well-recognized lesion."[1]

If these views are correct the old terms "optic neuritis," descending neuritis," and "papillitis," inasmuch as they give rise to erroneous impressions, should be abandoned. Nor is "tumor papillitis," suggested by Mr. Marcus Gunn, an improvement. Perhaps "choked disk" (staaungspapille) more nearly describes the process which actually exists than any other term except the one introduced by Elschnig, which is also recommended by Herbert Parsons, namely, "papillo-edema," and this term, we believe, at present gives the greatest satisfaction. It has been adopted, provided it is recognized as including all stages of the process, by Bordley and Cushing in their most recent communication on this subject.[2]

I. CONCERNING THE OPHTHALMOSCOPIC APPEARANCES OF THE NERVE HEAD WHICH INDICATE OPERATIVE INTERFERENCE.

Systematic writers have divided papillo-edema or choked disk into various stages, and for the purposes of the present discussion we may in part quote, with some modification, the descriptions of Mr. Marcus Gunn.[3]

(*a*) Increased redness of the disk, loss of definition of its edges, slight prominence of its surface, beginning filling in of the porus opticus represent the first stage.

(*b*) Edema of the nerve head, disappearance of the porus opticus, complete obscuration of the disk margins, moderate haze of

[1] Transactions of the Section on Ophthalmology, Jour. Amer. Med. Assoc., 1908.
[2] Ibid., p. 510.
[3] British Medical Journal, October 26, 1907.

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the surrounding retina and uneven distention and darkening of the retinal veins represent the second stage.

(*c*) Decided increase of the edema, elevation and size of the nerve head, striæ of edema, in the form of lines in the swollen retina between the disk and macula, marked distention of the retinal veins and retinal hemorrhages represent the third stage.

(*d*) Increase in the prominence of the disk, which assumes a mound shape and begins to lose its reddish color and to become opaque, exudation in and on the swollen disk and surrounding retina, elaboration of the retinal hemorrhages in size and number represent the fourth stage.

(*e*) Decided subsidence of the vascularity of the papillo-edema and increasing pallor, with or without sinking of its prominence, shrinking of the retinal arteries and thickening of their perivascular lymph sheaths, spots of degeneration in the retina, especially in the macula, represent the fifth stage, which soon passes into the final stage of so-called postpapillitic atrophy.

Vision, other things being equal, is usually good and capable of being preserved or improved by operation during the first, second, and third stages. Vision rapidly declines during the fourth stage, is very poor and sometimes obliterated in the fifth stage, and in either of these stages the prognosis *quoad visum* is most unfavorable, although not always, at least in the fourth stage, entirely hopeless.

Because so much of the success of operation in its relation to the preservation of sight depends upon its early performance, as will presently be shown, it is important to discuss for a moment whether (*a*) there are any sure signs of beginning papillo-edema before the symptoms appear which are present in the first stage, and (*b*) whether an early period of disk edema may be masked by other conditions.

Antedating the symptoms which indicate the first stage of papillo-edema, there may be the so-called imminence of neuritis or edema, in which the retinal vessels, often only one division of them, are darker, more distended, and more unevenly tortuous than is normal. Such a condition of affairs may possibly be due to pressure

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upon the vein in the intervaginal space which is already being dilated, and which is the place where, at the height of choked disk, this flattening of the vessel, as Dupuy-Dutemps has shown, reaches its maximum. It is not safe to make a decision at this stage by ophthalmoscopic examination alone. Whether carefully conducted photometric examinations, which usually reveal disturbances of the light sense at this period, would be of practical avail or not we are unable to say. Thus far they do not seem to have been very satisfactory. Of importance, however, is the field of vision, not the general field alone, but the effect of these early changes on the relation of the color lines one to the other. In this connection the investigations at present being made by Bordley and Cushing promise interesting results, and they intend to record them in the near future. The change in the size of the blind spot, particularly in the vertical direction, which has been found by Ramsay and Sutherland to be an early sign of congestion of the disk in sympathetic ophthalmia, and which depends upon turgescence of the superior and inferior branches of the retinal vessels, may possibly be a sign of early disk edema due to increased intracranial pressure. Concerning this symptom we hope to report at a future meeting of the College.

That an early stage of papillo-edema may be masked by a corresponding general retinal edema is an observation of great interest which has been made by Bordley and Cushing. In one of their cases both disk and retina were uniformly edematous, although the structures remained clear, and it was only after palliative exploration with decompression that the undetected retinal edema subsided in such a manner that the disks at once became prominent, which in their turn subsequently also regained their normal appearance. This observation we can confirm, as follows:

Case I.—A boy, aged nine years, with a bullet in his brain, was admitted to the surgical ward in the service of Dr. Frazier in the University Hospital, April 6, 1907, and an ophthalmoscopic examination made three days later records the absence of papillo-edema, although there appeared to be an ill-defined edema of the retina. On the 13th of the same month a decompressing trephining with exploration was performed by Dr. Frazier, and within a week,

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with the disappearance of the retinal edema, a marked papillo-edema, as much as 5 D., was evident. Following a second operation in which the bone was curetted for better drainage, this choked disk, much more marked in the right eye, began to disappear, and a month later had decidedly lessened, and we understand at the present time has disappeared, with the preservation of normal vision.

It would therefore seem, as Bordley and Cushing remark, that sometimes, without decompression, an incipient choked disk is extremely hard to distinguish from a simple hyperemia.

It is also difficult to differentiate a true congestion with edema, or beginning papillo-edema, from a spurious optic neuritis, or so-called hyperopic disk, as it occurs in association with refractive error. There is, however, in the beginning of papillo-edema, that is to say, when it has reached the first stage, a certain quality which pseudoneuritis does not possess. The blurring of the disk edges is more complete, there is evident contraction or filling in of the physiological pit, and even with the indirect method of examination, the obscured margins are with difficulty seen, not to mention the uneven tortuosity of the retinal veins, which has already been referred to. Here, again, a careful investigation of the size of the blind spot and of the light sense will probably lend aid in diagnosis.

II. CONCERNING THE DATE OF OCCURRENCE AND CHARACTER OF THE NERVE-HEAD CHANGES, TOGETHER WITH THE VISION.

It is not possible to predict how soon after a tumor develops changes in the nerve head will begin to appear. Sometimes, as Bruns has pointed out, the whole process from the beginning of papillo-edema to the height of its swelling is complete in a few weeks; sometimes months may elapse. In one patient with whose clinical history we are acquainted, the choked disk was postponed until within a week of the patient's death, although all symptoms of brain tumor had been present for more than a year. In general terms it may be stated that, with certain notable exceptions, the process must have existed for some time and the increased intra-

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cranial tension have lasted for a definite period before the engorgement edema develops.

While it is true that tumors of the cerebellum are prone to give rise quickly to disk edema, this is not always the case, and one record shows that a cerebellopontile tumor was present for eight years before papillo-edema developed (Bordley and Cushing).

In the experimental work of Bordley and Cushing, particularly the introduction of fluid into the subdural space, disk edema occurred very rapidly, indeed while the injection was taking place.

It is not possible, however, to determine from the stage of the retinal process what the duration of the cerebral lesion is, nor can we predict in the case of tumor in the absence of choked disk when this is likely to occur. We do know that disk edema may arise with great suddenness, and under such circumstances it indicates an increase in the intracranial pressure, either as the result of an elaboration of the growth itself, or on account of hemorrhage. To quote again from Bordley and Cushing, "The whole question resolves itself into a study of the character of the intracranial pressure and its effect upon the cerebrospinal circulation, regardless of the histology, the situation, or the duration of the neoplasm."

It is almost useless to point out again that choked disk caused by intracranial growths is perfectly compatible with good acuteness of vision, and that this may exist for long periods of time, although there is well-marked engorgement of the nerve head and all the signs of papillo-edema. Even when the visual acuteness appears not to be normal, but is reduced, for example, to one-half, or one-third, or even one-tenth of normal, it may sometimes be raised nearly or quite to the normal standard if any existing optical defect is corrected and, therefore, visual acuteness recorded without note of the refractive error and the effect of neutralizing lenses does not give a true indication of the real sharpness of sight. The important point is that good vision must not be permitted to stay the hand in operative interference, because what is good vision today may in a few days be poor vision, and already those processes may have started which, if unchecked, lead to the degenerations of the nerve, which ultimately end in blindness and atrophy.

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Whether the character of the refractive error bears any relationship to the rate of rapidity of the development of papillo-edema is somewhat in dispute. Touching this point, Mr. Marcus Gunn says: "Tumor papillitis is most commonly associated with hypermetropia, even in countries where this form of eyeball is not so common as in England, and papillitis is relatively rare in myopia, both here and abroad. It would appear as if in myopia an increased pressure within the sheath space was less likely to press directly upon the nerve and more likely to be relieved by filtration or absorption of the contained excess of fluid." This matter is also referred to by Paton in his well-known analyses of the effects of decompressing trephining on the preservation of vision, but Parsons, apparently quoting him, says, "There is no reason to suppose that myopia has any deterrent effect upon the development of papillo-edema, as has been thought," and Bordley and Cushing are evidently of the same opinion. We are not prepared, on account of insufficient experience, to express an authoritative opinion, but as far as it has gone it tends to show that if the patient's eye is myopic there is more apt to be a late development of papillo-edema than if the patient's eye is hyperopic, and in this sense our observations coincide with those of Mr. Gunn.

With these preliminary observations, we pass directly to the practical side of the present communication. For this purpose we have analyzed 21 cases of cerebellar tumor or cyst, 19 of which were operated on by Dr. Frazier in the University Hospital, and 2 of which were operated on in the Orthopedic Hospital, the one by Dr. William J. Taylor, and the other by Dr. G. G. Davis, both of these patients being in the service of Dr. Sinkler; and 22 cases of cerebral tumor, cyst, or abscess, 20 of which were operated on by Dr. Frazier in the University Hospital and 2 in the Orthopedic Hospital by Dr. William J. Taylor in the service of Drs. Lewis and Sinkler. Inasmuch as these cases have been utilized in other analyses from the surgical and neurological standpoints, we omit detailed descriptions and statistics and submit certain propositions and illustrative cases.

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1. *The effect of decompressing trephining, or of radical operation, on the preservation or restoration of eyesight: (a) when the vision is good in both eyes prior to the operation; (b) when the vision is good in one eye but practically lost in the other eye prior to operation; and (c) when the vision is defective, that is, below one-half of normal in both eyes.*

CASE II.—A girl, aged fourteen years, was admitted to the service of Drs. Spiller and Frazier in the University Hospital on June 24, 1908, with a diagnosis of encephalitis, or possibly of brain tumor, and with double beginning papillo-edema, the swelling of the disks being 1½ D.; vision with test types normal in each eye. Craniotomy, with opening of the dura, was performed on July 13, 1908, by Dr. Frazier. Six days later, vision still being normal, there was only slight edema, and seventeen days after operation the pupils were normal, there was no swelling of the disks, and only the slightest blurring of the nasal margins, vision being practically normal; that is to say, fully $\frac{6}{7.5}$ and probably better, as the patient was a little difficult to examine. So far as we are aware the good result remains at the present time.

CASE III.—A man, aged twenty years, referred to the University Hospital October 25, 1905, by Dr. Weisenburg, with cerebellar tumor, had papillo-edema of the right eye of 11 D. and a vision of $\frac{0}{9}$, and subsiding papillo-edema of the left eye with a vision of only shadows. Simple decompression was performed by Dr. Frazier without opening of the dura, and the first subsidence in the swelling of the edematous disk of the right eye was noticeable twelve days after operation. At the expiration of two months the papillo-edema had disappeared and the vision was $\frac{6}{9}$ in the right eye, while the vision of the left eye had risen to counting fingers. In this eye there was temporarily an increase of 1 D. in the swelling of the disk, which later slowly subsided. Five months later a second decompressing trephining upon the opposite side of the skull was performed by Dr. Frazier because of some increase in the patient's general symptoms, the vision of the right eye remaining $\frac{6}{9}$ as before, but the left, which showed increasing atrophy of the disk, had dropped again to shadows. The patient has remained well, and at the last report, two years after operation, he was performing his duties as a workingman each day.

It cannot be doubted that had the decompressing trephining not been performed, the right disk would have met the same fate which befell the left one.

CASE IV.—A man, aged twenty-three years, was admitted to the University Hospital December 12, 1903, with cerebellar tumor and a papillo-edema of the right eye of 4 D., and in the left eye slightly less; he had very poor vision, which unfortunately was not recorded with test types at the time of his admission,

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but which is reported to have been only the ability to see large objects. A decompressing operation was performed by Dr. Frazier, with removal of a portion of the cerebellar lobe, and at the expiration of seven days the vision had risen to $\frac{6}{15}$ in the right eye and $\frac{6}{7.5}$ in the left eye, and the edema of the disk had dropped 1 D. on the right side and 2 D. on the left. At the expiration of eleven weeks after the operation the vision of the right eye had risen to $\frac{6}{6}$, and of the left to $\frac{6}{6}$. In the right eye there was only a slight surface edema, and the left eye was normal. Four years after operation both disks were normal, and the vision of the right eye was $\frac{5}{7.5}$, and the left eye $\frac{5}{5}$.

The two cases which follow illustrate the good results along these lines of radical operations:

CASE V.—A woman, aged forty-five years, with cerebral cyst, had, just prior to the operation, a papillo-edema of 5 D. in the right eye and 3½ D. in the left eye, and a vision of $\frac{6}{15}$ in the right and $\frac{6}{7.5}$ in the left. The patient was admitted to the University Hospital February 5, 1908, in the service of Drs. Mills and Frazier and a large cerebral cyst removed, with complete disappearance of the edema and restoration of full visual acuity at the end of two months. Four months later the edema began again to appear, although vision continued to be normal. When the height of the swelling had reached 5 D. the brain was reopened and a large quantity of growth removed from the site of the original operation, followed, in the course of two months, by complete second disappearance of the papillo-edema, with a vision of $\frac{6}{6}$ or normal in the right eye, and $\frac{6}{7.5}$ in the left, on which side there were some indications of pallor in the deeper layers of the disk. This vision continues to the present time, the last test having been made a few days ago.

CASE VI.—A woman, aged twenty-three years, a patient of Dr. Morris Lewis in the Orthopedic Hospital, with cerebral tumor, had a double choked disk of 5½ D. in each eye and vision of $\frac{6}{6}$. A radical operation was performed by Dr. William J. Taylor over the fissure of Rolando, and the tumor removed. Rapid subsidence of the disk edema began on the second day and was nearly complete at the end of the seventeenth day, and the patient is now able to walk around and attend to her affairs; her vision is normal and the disk entirely recovered, nearly a year after operation.

All of these patients were operated on in the first or not later than the second stage of papillo-edema except one in whom the stage of the edema in the one eye had passed to the third period, and in the left eye was well advanced in the fourth or beginning of the fifth period, and the result was, as before noted, a preservation of

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the sight of the better eye. It is unnecessary to consume more time by the recitation of other cases. They serve to illustrate the first of the propositions presented this evening, and confirm Marcus Gunn's statement, as well as the experience of many operators, that if the intracranial tension is relieved in the first, second, or third stages of papillo-edema, the prognosis as to vision is decidedly favorable.

2. *To illustrate the danger of delay in resorting to operative interference, and the rapidity with which vision may deteriorate in cases of papillo-edema, which deterioration is checked by a suitable operation:*

CASE VII.—A male, aged twenty-five years, came to the University Hospital on January 29, 1906, with all of the symptoms of brain tumor and double papillo-edema, the elevation on the right side being 5 D., and on the left side 4 D. The vision of the right eye was $\frac{6}{7.5}$, and of the left eye $\frac{6}{9}$. Operation was urged and declined, and it was not until two months after his first examination that surgical interference could be undertaken. He was under constant medical treatment, but during the first twenty-six days of his treatment his vision fell to $\frac{6}{45}$ in each eye without any material increase in the size of the choked disk or change in the ophthalmoscopic appearances. Decompressing trephining was now performed by Dr. Frazier, with opening of the dura. Within five days the disk edema began to subside, first in the right eye. It had practically disappeared by the end of the second month, and at the last examination, eight months after the operation, the vision of the right eye prior to operation was maintained at $\frac{6}{45}$, while that of the left eye had improved from $\frac{6}{45}$ to $\frac{6}{15}$, in spite of some evidence of beginning atrophy. So far as we know this vision is maintained at the present time, now more than two years after the operation.

It cannot be doubted that had it been possible to perform the operation when the patient first entered the hospital, that is to say, when his vision was $\frac{6}{7.5}$ and $\frac{6}{9}$, this acuteness of sight would have been preserved, because the depreciation of vision which so rapidly manifested itself was immediately checked by the trephining, and ultimately, in so far as the left eye is concerned, improvement in visual acuteness to the extent already recorded was evident.

The outcome, however, is not so fortunate as this in all cases if the patient declines operative interference.

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CASE VIII.—A woman, aged twenty years, was first examined by one of us November 22, 1906, with all of the symptoms of brain tumor and double papillo-edema of 5 D. in the second stage of this process, but with normal visual acuteness, that is to say, $\frac{6}{6}$ in each eye. In spite of frequent urging to submit to operation, she went on with the usual treatment of iodide and mercury. Exactly when the vision began to fail we cannot state, as she passed from our own observation and we did not see her again until she became a patient in the service of Drs. Mills and Frazier in the University Hospital, January 9, 1908, or thirteen months after the original examination. Papillo-edema in the late fourth or the beginning fifth stage was present, and vision was reduced to the perception of very large letters held quite close to the eye. A decompressing trephining was performed, with relief of the general symptoms in so far as we are aware, but without any effect in restoring or preserving the declining vision.

This experience could be emphasized by the recitation of a number of cases, which would only unnecessarily consume time. Inasmuch as the operation was an entire success from the surgical standpoint at the time it was performed, it cannot be doubted that an equal success would have occurred had the trephining been possible when the vision was still $\frac{6}{6}$, at which standard it doubtless would have been maintained.

3. *To illustrate the preservation of, or even slight improvement in, very poor vision existing prior to the operation by a decompressing trephining.*

A question of importance to decide in so far as vision is concerned is this: Shall the surgeon operate even though the papillo-edema has reached the fourth or even the fifth stage? Certainly even in the fourth stage the prognosis *quoad visum* after operation is unfavorable, and so high an authority as Mr. Gunn states that if the fifth stage is reached there is no reason, so far as sight is concerned, for performing any operation; but he is willing that the operation shall be performed in the fourth stage, because he has known useful vision to be obtained after operation even at this period.

CASE IX.—M. B., a woman, aged twenty-nine years, with cerebral tumor, had papillo-edema in the fourth stage of 3 D. in the right eye and 4 D. in the left eye, in which, indeed, the signs of the fifth stage were already beginning to be

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manifest. The vision of the right eye was $\frac{1/2}{150}$; the left eye was totally blind. A decompressing trephining was performed by Dr. Frazier, and at the expiration of ten months after the operation, at which time the last ocular examination was made, the vision of the right eye had improved from $\frac{1/2}{150}$ to $\frac{1/2}{50}$; all swelling of the disks had long since disappeared.

CASE X.—A male, aged fifteen years, with cerebellar tumor, blind in the right eye and with a vision of $\frac{1}{100}$ in the left eye, had double papillo-edema in the late fourth or beginning fifth stage of 6 D. in the right eye and 4 D. in the left eye. Decompressing trephining, with opening of the dura, was performed by Dr. Frazier, and the first signs of subsidence of the choked disk were manifest on the nineteenth day, and at the expiration of two months almost all of the swelling had disappeared, and the vision of the left, or only eye which had any sight prior to the operation, was still preserved, in fact was slightly better, being $\frac{1}{60}$, as compared with $\frac{1}{100}$.

Certainly, these two cases are good illustrations of our contention and also the contention of Mr. Gunn, that even though the visual prognosis is unfavorable, and a late stage of the papillo-edema has been reached, blindness may still be averted and a decompressing trephining should be tried.

Unfortunately this is not always the case, as is illustrated by the following record:

CASE XI.—A girl, aged seventeen years, with cerebellar tumor, had a papillo-edema of 9 D. in each eye in the late fourth or beginning fifth stage, with a vision in the right eye of $\frac{2}{60}$, the left eye being totally blind. Decompressing trephining, with opening of the dura, was performed by Dr. Frazier, and already, on the fourth day, there was a subsidence of 3 D., and at the expiration of a month a practical disappearance of the papillo-edema, without, however, preserving the slight vision which had existed prior to the operation, inasmuch as the $\frac{2}{60}$ at that time was reduced to hand movements, and a short time afterward disappeared entirely, the blindness being absolute. There could not be a better illustration of the misfortune of delayed operation.

All who have to do with the blind will realize how much it means to the sufferer if he has only light perception, and how he will cling to that small blessing with almost the same tenacity with which we would cling to the preservation of good vision. Therefore it would seem that even from this standpoint it is fair to give the patient a chance by a decompressing trephining,

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although there is no hope of restoring vision, but only hope of preserving this faint light perception. The following is a good illustration:

CASE XII.—A girl, aged fourteen years, with double papillo-edema of 5 D. in a late state of its development and vision reduced to faint light perception, had a decompressing trephining with opening of the dura performed by Dr. Frazier. Already at the end of the first day there was distinct subsidence of the swelling, and one month later it had absolutely disappeared, fortunately leaving the patient with the light perception that existed prior to the operation, but without improvement.

It cannot be doubted that had the operation not been performed, the small measure of sight which this patient possessed would have been speedily totally lost. Whether what she now has will later disappear cannot be stated, but at least for a period of time she has had this satisfaction, and, as we have said, to a blind person it is not a small one.

On the other hand, in a certain number of cases of brain tumor, with very little vision prior to the trephining, for example, a vision reduced to hand movements or even light perception only, this sight may rapidly disappear after the trephining, especially if there has been much hemorrhage. For example, three patients, the one with a tumor of the lobe of the cerebellum, another with a growth of the cerebellopontile angle, and the third with a cyst of the left lobe of the cerebellum and with much reduced vision prior to the operation—namely, hand movements in two and the ability to distinguish very large letters held close to the eye in the third—were completely blind, even to the abolition of light perception within the first week after operation, although in each instance the surgical success was a brilliant one. We do not think, however, that experiences of this character should be recorded as contra-indications for the operation, because these patients with very much reduced vision are doomed to speedy blindness without operation, and with it they have a small chance of the preservation of such sight as they have.

We come now to take up certain complications and disappointments connected with the operation which is under discussion.

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1. *The occasional delay in the subsidence of the papillo-edema and the restoration of vision, but with an ultimate good result.*

CASE XIII.—A woman, aged fifty-one years, with a tumor of the cerebello-pontile angle, had a papillo-edema of 4 D. in the right eye and 3 D. in the left eye, and a vision of $\frac{6}{9}$ in the right and $\frac{6}{9}$ in the left. After decompressing with opening of the dura, there was absolutely no change in the appearance of the disks at the expiration of several weeks. After a second operation performed by Dr. Frazier, in which the dura was opened and some blood evacuated and the dura left unsewed, there was again no change for nearly a month, but on the twenty-third day the disk edema began to subside, and five weeks later the swelling of the disks had been reduced to 2 D. in the one and to 1½ D. in the other eye, and vision had improved in the right eye from $\frac{6}{9}$ to $\frac{6}{6}$, and was practically maintained in the left eye at $\frac{6}{7.5}$.

This case illustrates a point to which Mr. Gunn also makes reference when he says: "Visual improvement after operation, although long delayed, may ultimately be very satisfactory."

2. *Increase in papillo-edema and visual disturbance following decompressing trephining.*

Everyone interested in this matter must have noticed in a certain number of cases a temporary depreciation of vision within the first week after operation, probably due to shock, perhaps to hemorrhage, and which is apparently of no importance in the subsequent restoration or preservation of vision, provided the primary vision has been good and the disk edema of comparatively short duration. So, too, in other cases during the first day or two after trephining, there may be a slight increase in edema associated with fresh hemorrhages. This is apparently of no importance, as the added edema and fresh hemorrhage disappear in the subsequent general subsidence of the swelling. For example:

CASE XV.—A girl, aged seventeen years, with sarcoma of the brain and double papillo-edema of 5 D. each, had a decompressing trephining performed by Dr. Frazier, with opening of the dura. At the end of eleven days vision had decreased and the papillo-edema increased 2 D. on the right side and 1 D. on the left, but one month later there was gradual improvement, followed by the usual subsidence.

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This temporary increase of edema is probably due, as Bordley and Cushing point out, to manipulation of the brain at the time of operation, or sometimes depends upon hemorrhage which has gathered beneath the dura and which must be let out at a subsequent operation. The point is that if it occurs the prognosis should not be too unfavorable and it should lead to a careful examination of the field of operation to ascertain whether some such complication as has been referred to has taken place.

3. *Preservation of the vision of one eye, but failure to preserve the sight in the opposite eye by a decompressing trephining.*

CASE XVI.—A woman, aged twenty-one years, with cerebral tumor, had a vision of $\frac{6}{6}$ in each eye and a papillo-edema in the third stage of 7 D. in each eye. A decompressing trephining, with opening of the dura, was performed by Dr. Frazier, and at the expiration of nine days there was marked subsidence of the swelling, and after one month it had been reduced just 50 per cent., with complete preservation of the vision of the right eye, but with a reduction of the vision of the left eye from $\frac{6}{6}$ to counting fingers.

It is possible that under these circumstances in the eye which fails to regain or preserve its vision after the trephining, atrophy of the nerve has already begun, but has not yet become ophthalmoscopically manifest, and is usually the eye on the same side as that on which the lesion occurs. In other words, the degree of the swelling of the papilla is not always an accurate indication of the visual prognosis, nor is the ophthalmoscopic examination always a complete guide as to what process has already started deeper in the nerve.

4. *Loss of vision which had been good prior to the operation and with the papillo-edema still in an early stage.*

We have already seen that the prognosis as to vision after decompressing trephining is unfavorable when the disk edema is at a late stage of its development, and that though occasionally successful in preserving what vision exists, it is sometimes also followed by rapid disappearance of the trifling vision which the patient has. Unfortunately, also, occasionally after technically correct operations, when one would expect good results, there is rapid loss of vision.

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CASE XVII.—A boy, aged ten years, a patient of Dr. Sinkler's, in the Orthopedic Hospital, with cerebellar cyst and double papillo-edema of 8 D. in the right eye and 7 D. in the left eye, had a vision of $\frac{6}{15}$ in the right eye and $\frac{6}{8}$ in the left eye. Trephining was performed by Dr. William J. Taylor, and the operation suspended on account of severe hemorrhage. At the expiration of thirteen days there was not the slightest change in the swelling of the disks. Twenty days after the first operation a second one was performed, the dura opened, and the cyst ruptured during the manipulation, with the result that at the end of three days the swollen disks had been reduced respectively to 3 D. and 5 D., and at the expiration of a little more than a month their swelling had entirely disappeared; but in the meantime rapid atrophy had ensued, and the right eye had become totally blind and the vision of the left eye reduced to $\frac{6}{60}$ with difficulty. The last examination, made a few days ago, indicates slight visual improvement, namely, $\frac{6}{15}$.

Referring to these cases, and there are not a few of them on record, one of us has written as follows: "Occasionally there is a rapid loss of sight after trephining when one would expect just the contrary result. A possible explanation of this unfortunate sequel is great loss of blood at the time of operation. The hemorrhage may have caused some change in the ganglion cells of the retina, exactly as such changes may be produced by free loss of blood after hemorrhage from the stomach or bowels. If so, the mechanism of the blindness under these circumstances is the same as that which occurs in quinine and in methyl-alcohol poisoning."

Concerning these cases, Dr. Harvey Cushing[1] writes as follows: "I recall very much the same experience that you speak of on one or two occasions, and it is possible that there may have been others. It is my impression, however, that they have all been patients in whom just before the time of operation there had been rather a rapid loss of visual acuity. I think I may possibly be able to explain the loss of vision which occurs in some cases of subtentorial lesion, even though the swelling may subside and even though vision may have been fairly good at the time of operation. Total blindness, however, with total deafness, occurred in two of my patients shortly after suboccipital explorations, which disclosed no lesion. Both of these patients had a growth involving

[1] Personal communication.

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the corpora quadrigemina, and the blindness and deafness were attributed to implication of the geniculate bodies. Is it possible that a similar condition could have been instrumental in the loss of vision in the patient with cerebellar cyst whom you mention?" He also discusses the relationship of acute obstructive hydrops ventriculorum, which is present in subtentorial lesions, and which, as we know, leads to a high grade of papillo-edema and subsequent atrophy. In these cases of cerebellar cyst he says that there may be a gradual shutting down upon the nerve of the new-formed connective tissue during the process of subsidence of the swelling.

In conclusion, we desire to call attention to certain practical points connected with the ophthalmoscopic examination of patients suffering from intracranial tumor, both with and without decompressing trephining.

1. *The earliest date at which subsidence of the swollen disk is noticeable.*

In eleven cerebellar tumors recently observed, the first distinct lessening in the disk edema after operation, that is to say, at least 1 D. was noticed as early as the end of the first day in one case and deferred as late as the twenty-third day in another case, the average being a little over nine days. A similar analysis of nine cerebral tumors showed that the first decrease occurred in one case as early as the end of the second day, and in another case, in which there was a primary increase of the edema, it was delayed to the thirtieth day, the average being a little over ten days. A very common result, even in those cases in which the disk edema itself does not appreciably diminish for the first few days, is a lessening of the general vascularity of the eye-ground, which is often quite noticeable within a few hours after the trephining, although we have not been able to observe it while watching the eye-ground during the progress of the operation. This, however, is rather difficult to do, and doubtless future investigations along these lines may yield other results. In general terms, it may be stated that while the disk edema may subside as much as 1 D. within the first twenty-four or forty-eight hours, and quite commonly begins to subside from the third to the fourth day, an

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average of a number of cases usually indicates that the first distinct improvement occurs from the ninth to the tenth day. This observation is in accord with the statistics already published by Mr. Paton, who states that generally within a week or a fortnight after the second operation (all of his cases were submitted to two operations; or, rather, to two stages of operation, the first consisting in removal of the bone and sometimes incision of the dura, and the second, if possible, the removal of the tumor itself) there was distinct subsidence of the swelling, but between the two operations there was little measurable alteration in the disks.

According to Mr. Gunn the date of the first occurrence of the subsidence of disk swelling and the rapidity with which it proceeds vary considerably, but are earlier and more rapid in cases where the operation has been performed not later than the third stage of disk edema. We have not found in our observations that there was any material advantage, in so far as the preservation or restoration of vision is concerned, in sudden subsidence of disk edema, or, more accurately, in rapid subsidence, with perhaps one notable exception, operated on at the Orthopedic Hospital by Dr. Taylor. Indeed, we have come to believe that the more gradual subsidence, beginning at about the tenth day, yielded more favorable results than when the disk elevation subsided more rapidly. This point, however, is one which would require further study before it could be presented in a dogmatic statement.

2. *The indications which the disk appearances give as to the size and situation of the growth; that is to say, whether it is on the right or left side of the brain or cerebellum.*

As we have already stated, and as has been pointed out many times by other observers, ophthalmoscopic examination yields no information in regard to the size or structure of a tumor, because it is perfectly well known that small growths may cause an elevation of intracranial pressure greater than large and infiltrating ones, depending upon the situation of each. If the growth has caused an increase in intracranial tension through what has been called the intermediation of an internal hydrocephalus, as Bordley and Cushing point out, the disk edema is apt to be equal in the two eyes, and

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it is probable, as these authors further maintain, that if every brain tumor exerted its pressure equally in all directions the swelling in the two disks would always be likewise equal. This we know is not always the case; and, moreover, under certain circumstances there is a greater pressure of fluid in the optic nerve sheath on one side than the other, and therefore a greater swelling of the optic disk in one eye than the other. Whether this greater swelling on one side is a sufficient indication that the growth is also upon the same side of the intracranial contents is a matter of dispute. Thus, Sir Victor Horsley holds that the disk edema, or neuritis as he calls it, occurs first of all, or is more intense, on the side of the tumor, while Mr. Leslie Paton's observations indicate that no reliance can be placed on this sign, for although the preponderance was somewhat in favor of the more severe neuritis being on the affected side, yet in some cases it was more marked on the opposite side. On the other hand, Sir William Gowers, and apparently Dr. Beevor, agree with Horsley in regard to the homolaterality of the disk swelling and the tumor. Touching this point, Bordley and Cushing state that in 70 per cent. of their cases the papilla on the side of the tumor was the most involved, and in 80 per cent. of the cases seen before the choked disk made its appearance the first swelling ultimately occurred in the eye homolateral to the tumor, and in 72 per cent. the ultimate damage to the nerve was greatest on this side.

So far as we are able to judge from our own experience, we would say that in the majority of cases the greater swelling is on the same side as the tumor, but we would agree with Horsley, Cushing, and others, that it is not so much to the swelling of the disk that attention should be paid, as to the indications as to which disk has been longest affected by finding the evidences of beginning atrophy, by attention to which side of the disk has first been affected, the nasal or the inferior quadrant, and by the information which is given by a test of the visual acuteness, the light sense, and the field of vision. As Bordley and Cushing point out, a tumor may, for instance, be present on one side of the brain, and by the character of its growth impinge on the opposite side and

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cause an unequal forcing of cerebrospinal fluid into the intravaginal sheath of the optic nerve, and therefore a greater swelling of the disk on the opposite side of the tumor; or it may happen that the examination is made when the disk which was originally the largest in size had already begun to decline, and this would throw an element of doubt on the examination. Therefore, the mere swelling alone does not furnish the most important information, but the other symptoms, to which we have called attention. So sure of this is Sir Victor Horsley that he states: "Within my own knowledge the early failure of visual acuity indicates beyond doubt the situation of the tumor." The essential point, according to him, is the estimation of the age of the neuritis, and this means minute examination of the interstitial tissue of the disk.

3. *Delay in the development of the optic disk changes.*

We have already pointed out that there is some reason to believe that a study of the refraction of the eye is of some importance in the determination of this point, and that although all observers are not in entire accord, there is a good deal of evidence to show that in the presence of myopia the disk changes do not develop as quickly as when the other type of refraction is present.

A still more interesting question in this connection is the age of the patient, and if Singer's tables are correct, the absence of optic neuritis, or disk edema, in intracranial tumors, exclusive of those occurring in the pons, is rare in cases under forty years of age and becomes increasingly more frequent after that period of life. Thus in an analysis of 88 cases of cerebral and cerebellar tumors, he found in 35 cases under thirty years of age well-marked choked disk in 97.1 per cent. of the cases, and its late or slight development in only 2.9 per cent. of the cases, it being absent in none of them. Between thirty and forty the figures read thus: 18 cases, well-marked neuritis, 76.6 per cent.; late or slight neuritis, 2.2 per cent.; absent, 1.1 per cent.; between forty and fifty the neuritis was well-marked in 61.9 per cent. of the cases, 21 in all, and absent in 19; while over sixty years of age in 7 cases it was not well-marked in any of them, late or slight in 14.3 per cent. of them, and absent in 57.1 per cent.

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If these figures are correct, and we shall return to more elaborate analyses at some future time, the search for the earliest stages of papillo-edema, according to the directions already given, in cases of suspected brain tumor after the thirtieth year of life, becomes increasingly important.

In final conclusion we would say:

1. The most satisfactory treatment for the purpose of preserving vision in any case of choked disk or papillo-edema not due to a toxic process or constitutional disease ("infectious optic neuritis"), but depending upon increased intracranial tension, is decompressing trephining, with the removal of the growth if it is accessible.

2. This operation should be performed early, and if it can be done during the first, second, or even third stage of papillo-edema, the prognosis as to sight is most favorable.

3. If for any reason the operation is postponed until the development of the fourth and fifth stages of papillo-edema, already associated with marked depreciation of vision, the prognosis as to sight is unfavorable; but even under these circumstances the operation should be performed, because it sometimes preserves such vision as still remains, and if it should happen to be followed by a rapid depreciation of vision, as, unfortunately, is apt to occur, it at least gives the patients a chance, because without it they are doomed to blindness.

4. The investigation of the eyes under these circumstances must include not only an ophthalmoscopic examination, but one which includes a careful investigation of the visual field, the color perception, light sense, size of the blind spot, etc.

5. Patients afflicted with papillo-edema dependent upon increased intracranial tension should have the case fairly stated to them, and the operation should be urged in spite of occasional unfavorable results, because in its absence ultimate blindness is almost sure to result.

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DISCUSSION.

DR. CHARLES K. MILLS: It is more than twenty years since I first was able, with the help of the surgeons and neurologists, to see some advantage from trephining in the relief of such symptoms as headache and choked disk or papillo-edema. This relief occurred occasionally in cases where the operation failed to reveal the existence of a tumor which had been suspected and had been diagnosticated. These observations, however, were simply incidental or accidental in connection with procedures for the radical removal of growths.

A few years since, at the University Hospital in particular, we began to study more closely the effects of palliative or decompressive operations independently of the question of radical removal of brain tumors. Dr. Spiller, as Dr. de Schweinitz has pointed out, at a meeting at which I read a paper on some phase of this subject of brain tumor, spoke of this question of decompression for papillo-edema, and the matter was discussed at that meeting. Since that time, at the University Hospital in particular and elsewhere to some extent, we have frequently urged and with increasing energy as the result of experience, the resort to decompressing operations. I became satisfied some years since that these operations were imperatively demanded. I have been so much impressed with this idea that for the last few years I have been in the habit of urging operation not only as soon as papillo-edema was present, but when from the observation of one like Dr. de Schwenitz, on whom I could rely, it was imminent. Great mischief has often resulted from delay. Dr. de Schweinitz, in the systematic and orderly manner in which he has presented the subject which has been under discussion for several years, has called attention to this question of delay. I was one of the first to speak strongly against delays, against the administration of mercury and the iodides, waiting for their effects while the patient's sight was failing, and also waiting for a more accurate focal diagnosis or to more fully corroborate the general diagnosis.

I am not altogether in agreement with Sir Victor Horsley and others, if I understand the matter correctly, that the relative degree of the choking of the disk of one eye is of much value in focal diagnosis. My own experience has been that the papillo-edema has often been as great upon the side opposite to that on which the lesion is located as it has been on the same side; at any rate, it would be a mistake to rely upon a sign of this kind rather than upon other and better focal signs.

With regard to the rapid subsidence of the swelling after operation, my experience is fully in accord with that of Dr. de Schweinitz; in fact, I presume that his experience and mine are the same, as some of the cases observed have been cases with him in my own service or in private practice. I do not like to learn from the ophthalmologist that the swelling has very rapidly subsided after an operation. The cases are likely to turn out more successfully if this subsidence is postponed for six or eight days.

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DR. CHARLES H. FRAZIER: Dr. de Schweinitz has presented in a very comprehensive way the effects of so-called decompressive operations upon papillo-edema. It has been a very great satisfaction to me to have had the benefit of his advice, his experience, and his suggestions in determining the question of operation in many of my cases, and it is needless to say that the value of our operative records has been greatly enchanced by his personal contributions.

I should like to say a few words bearing directly upon the operation of decompression, because I believe that the profession at large has not a very clear conception of its precise nature. As the term implies, this operation is designed to relieve intracranial tension, not only in cases of tumors, but in chronic meningitis, with or without hydrocephalus, in the edema of cerebral traumatism and in epilepsy. When Annandale or Horsley first suggested, some fifteen years ago, the propriety of removing a portion of the skull as a palliative procedure in cases of brain tumor, the operation consisted solely in the removal of a button of bone with the conical trephine. Since that time the technique has been elaborated and a decompressive operation in the modern acceptance of the term implies the removal not only of an area of bone 3 or 4 cm. in diameter, but also of the removal of a corresponding portion of the dura, or at least a crucial incision in the dura. Sänger advocated the right temporal region as the site of election, because in this region one uncovers the slightest area of the brain, and Cushing proposed that the region be exposed by reflecting a flap of skin and temporal fascia by splitting the temporal muscle in the direction of its fibers. If I may be permitted to exhibit a few lantern slides, they will suffice to illustrate the distinction which I have attempted to bring out between purposeful exploratory craniotomy, an exploratory craniotomy followed by decompression, and purposeful decompression pure and simple.

As to the effects of the operation, Dr. de Schweinitz has already referred to its influence upon papillo-edema, and while the preservation of vision is one of the most urgent indications of decompression, its effect upon other conditions is a matter of no small consequence. I refer especially to headache, nausea and vomiting, and vertigo.

From an experience including between fifty and sixty decompressive operations I can say without any hesitation that I know of no form of therapy offering a greater measure of relief to such a hopeless and distressing class of cases. In many instances the relief afforded by the operation is immediate, and may continue for months if not years.

It might be of interest in this connection to speak of the influence of decompression upon the growth of the tumor. In a few cases the sudden relief of tension seemed to be followed by more rapid growth, and in others the growth seems to have been retarded. Whether or not in any, as Horsley firmly believes, the growth of the tumor has not only been arrested but undergone retrograde changes, must be a matter of conjecture. Suffice it to say that in one

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or two of our cases symptoms have not recurred after an operation of four or five years.

The importance of early operation has been dwelt upon by Dr. de Schweinitz. May I refer to it again, if only for the sake of emphasis. We have had too many cases, victims of the indefinitely prolonged courses of iodides and mercury. Six weeks or two months at most is long enough to establish a diagnosis. If the condition is not materially improved by this time, operation should be resorted to without delay, whether the lesion be a gumma or a neoplasm.

DR. WILLIAM J. TAYLOR: I agree thoroughly with Dr. de Schweinitz in regard to the necessity for early operation in all of these cases, and my own experience is that it is of the very greatest benefit. I wish to thank Dr. de Schweinitz very much for the way in which he has presented his facts, and I feel that I myself have gained a great deal in listening to them.

DR. WILLIAM G. SPILLER: I should like to ask Dr. de Schweinitz whether there is any way by which he can distinguish from ophthalmoscopic examination the papillo-edema of increased intracranial pressure. In a case of encephalitis under my care some time ago beginning papillo-edema was present, and I am not inclined to regard it as caused by increased intracranial pressure. We are not convinced that all papillo-edema is caused by increased intracranial pressure.

We are well aware of the great difficulty often encountered in trying to persuade patients to have an early decompression. I recall a recent patient, to whom Dr. de Schweinitz alludes, to whom I urged operation in vain. It is often impossible to make people with only slight disturbance of vision believe that they are in a serious condition. They may have none of the other signs of increased intracranial pressure. Edema of the disk not infrequently is absent in tumor of the motor region or of the pons, and I have known extensive glioma of the pons to be without papillo-edema a long time. The tumors of the cerebello-pontile angle often are fibromata, grow slowly, and cause atrophy of the surrounding brain tissue; and therefore may not produce early papillo-edema. Dr. de Schweinitz has not encouraged us to operate in the fifth stage, but aside from the impaired vision we have to bear in mind the other very serious symptoms. I recall a patient who was blind before the decompression, but relief from the other symptoms fully justified the operation. When the sight is lost after a decompression operation Dr. de Schweinitz has noticed that the loss is usually on the side of the tumor; inasmuch as this seems to indicate a greater intensity of the process on this side, it would appear to confirm Horsley's view that the nerve on the side of the tumor is more likely to become affected first.

Dr. de Schweinitz has brought out an interesting point in the increase of blindness occurring after subtentorial decompression, resulting from internal hydrocephalus. I should like to know whether he has observed blindness following decompression above the tentorium. Pronounced internal hydro-

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cephalus may occur with tumor of the cerebello-pontile angle. Decompression in the occipital region may, by drawing away the fluid of the posterior cranial fossa, cause the brain to be driven against the base of the skull and into the foramen magnum from the increased pressure above, which is not promptly relieved by the operation, and in this way increase the internal hydrocephalus and cause blindness.

DR. JOHN B. ROBERTS: Two things have struck me in this discussion, and they were suggested also by seeing the title of the paper. First we do not appreciate for a long time the collateral advantages of new operative procedures. Many of us have been doing new operations on the trunk and skull for various surgical conditions, but fail for a long time to see the collateral advantages of these operations. The second thing is the great value of coöperative clinical work by medical and surgical specialists.

DR. F. X. DERCUM: Many years ago I called attention to the fact that a growth in the posterior cerebellar fossa was frequently accompanied by a neuritis which was usually very rapid in development and intense in degree. I should like to ask Dr. de Schweinitz whether in cases of optic neuritis presenting such features it would not be advisable to recommend sub-tentorial decompression rather than decompression in the right temporal region.

DR. JOHN H. GIBBON: I should like to have Dr. de Schweinitz tell us something about the permanency of the relief. I have had only one case of pontine angle tumor. The woman, who was totally blind, did regain light perception, but this shortly after disappeared. I should like to know whether the improvement is permanent or whether it is lost before the patient succumbs to the disease, as most of them do. I do not mean by this interrogation to indicate that the improvement does not warrant the operation, even if it is temporary, but only ask for information.

DR. DE SCHWEINITZ: In answer to Dr. Gibbon's question as to the permanency of the results of decompressing trephining and the radical operation, I have to say that this varies very much. That patients may retain full vision for long periods of time is proved by one of our cases in which the operation was performed in 1903, and the man at the present time has practically normal sight. In a number of other cases the good results are still evident two and three years after the operation. As has been pointed out, however, in some cases, especially when there is very poor vision prior to the trephining, there is a rather rapid decline of sight, but even under such circumstances it seems to me proper to give these patients the chance which operation affords, even if it saves sight only for a comparatively short period of time.

To Dr. Dercum's question I would reply that it is more suitably answered by the surgeons, whose experience in these respects is of course much larger than my own. If I understand the matter correctly, operative treatment, in the absence of other indications, should consist in opening the subdural space in the temporal or subtentorial region, and that if a cerebellar tumor were

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suspected, the subtentorial operation is preferable, and that by its performance, other things being equal, a greater effect is secured. It is of course extremely difficult to state positively by an ophthalmoscopic examination that a so-called optic neuritis, or, as we prefer to call it, a papillo-edema, is always due to increased intracranial tension, but in most instances a reasonably good diagnosis can be made if the various symptoms which were outlined in the paper are carefully studied. I am not in the least desirous of being overenthusiastic about a serious surgical operation, but so far as my experience has gone, it corroborates that which has been quoted from other authors, that early decompressing trephining is at the present time the best method which we have of dealing with papillo-edema due to increased intracranial pressure, and that if the operation is to be done, then it should be done quickly and before those changes occur which vitiate its results because of the beginning of optic nerve atrophy.

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THE CAMMIDGE REACTION IN EXPERIMENTAL PANCREATITIS.[1]

By JOHN SPEESE, M.D.,

AND

EDWARD H. GOODMAN, M.D.

(From the William Pepper Clinical Laboratory of the University of Pennsylvania, and the Private Laboratory of Dr. J. H. Musser, Philadelphia.)

It is rather surprising that, despite the great clinical value claimed for his test, Cammidge[2] has so far made no attempt to control his observations by experimental work. Eichler[3] studied the effect of experimental pancreatitis on the urine, producing in one dog hemorrhagic pancreatitis, and in two dogs a diffuse purulent pancreatitis. The urines of these dogs before operation were negative, confirming the observation previously made that the urine of normal men, dogs, and rabbits does not give a positive pancreatic reaction. The postoperative examination of the urine in each instance was positive, which led Eichler to conclude that the test is of great value in diagnosticating pancreatic disease. As he used the *A* and *B* reactions in his studies, and as these reactions have been severely criticised by Ham and Cleland,[4] Schroeder,[5] Gruner,[6] Wilcox,[7] and Haldane,[8] the value of Eichler's conclusions may be questioned.

In 1907 Cammidge[9] described a third, or *C* reaction, which he

[1] Read December 2, 1908.
[2] Cammidge-Robson and Cammidge, The Pancreas: Its Surgery and Pathology, 1908.
[3] Berl. klin. Woch., 1907, p. 769.
[4] Australasian Medical Gazette, 1904, p. 399; Lancet, 1904, i, 1378.
[5] American Medicine, 1904, p. 406. [6] Lancet, 1904, i, 1459. [7] Ibid., ii, 211.
[8] Edinburgh Medical Journal, 1906, xx, 418. [9] Loc. cit., p. 252.

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claimed does away with many objections raised by his critics. Very little clinical work has been done with the *C* test, apart from Cammidge's own researches, and, so far, no experimental work has been reported. The papers of Cammidge, Schroeder,[1] and Goodman have presented so much clinical evidence in favor of the reaction that we deemed it advisable to control these observations by experimentation.

During a study of experimental pancreatitis conducted by Sailer and Speese,[2] the examination of the urine for the Cammidge reaction was productive of such interesting results that we were led to a systematic study of this phase of the question.

In five dogs 10 to 30 c.c. of cottonseed oil was injected into the pancreatic duct, a procedure which causes rapid necrosis of the pancreas. In three of these cases studies were made of the urine before and after operation, and in two cases the examination before operation was omitted. The dogs were killed eight hours after the operation.

CASE I.—The urine before operation was not examined. The urine after operation gave a positive reaction. Macroscopic examination of the pancreas showed a diffuse necrotic process which involved most of the organ. The microscopic study revealed complete destruction of the tissue.

CASE II.—The urine before operation was negative; the urine after operation was negative. The pathological examination of the pancreas showed neither macroscopic nor microscopic lesions. In this case pancreatitis was not produced, owing to the attempt to inject oil into the pancreatic duct being unsuccessful.

CASE III.—The urine before operation was not examined; the urine after operation gave a positive reaction. The postmortem examination of the pancreas showed a diffuse necrotic process; lesions similar in nature were seen microscopically.

CASE IV.—The urine before operation was negative; the urine after operation gave a positive reaction. The macroscopic and microscopic study of the pancreas showed a diffuse necrotic process.

CASE V.—The urine before operation was negative; the urine the first hour after operation was negative; the urine the fourth hour after operation gave a positive reaction. The usual necrotic appearance following oil injections was

[1] Journal of the American Medical Association, 1908, li, 837.
[2] Transactions of the Association of American Physicians, 1908.

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seen; the microscopic examination failed to reveal any normal structure except in the tail of the pancreas. The animal in this case was killed four hours after operation.

Encouraged by these results, we thought it of interest to study the urine in cases of non-acute pancreatitis. Ligation of the pancreatic duct seemed to be the simplest method of producing such a lesion. The plan which we adopted in three instances was to examine the urine before ligation of the duct, and at the end of each twenty-four hours, following the operation. After a positive reaction was obtained the animal was killed and the pancreas removed for microscopic study.

CASE VI.—The urine before operation was negative; the urine the first day after operation was negative; the urine the second day after operation gave a positive reaction; the urine the third day after operation gave a positive reaction; the urine the fourth day after operation gave a positive reaction; and the urine the fifth day after operation gave a positive reaction. The dog was killed seven days after operation. An area of induration was found in the head of the pancreas, opposite the point of ligation. This lesion was the size of a large walnut, was distinctly hard, and cut with greater resistance than the remaining portions of the gland. Microscopic study of the indurated tissue showed a subacute inflammatory process, which had caused small areas of necrosis in the acini. A leukocytic infiltration, consisting for the most part of round cells, was seen in the stroma, and, to a less extent, about the acini. Proliferated connective-tissue cells were also noted.

CASE VII.—The urine before operation was negative; the urine the first day after operation gave a positive reaction; and the urine the second day after operation gave a positive reaction. The dog was killed on the second day. The pancreas was indurated in an area about the size and shape of a hickory nut, the lesion being directly above the ligature which still surrounded the duct. Microscopic examination showed changes similar to Case VI, but not so extensive.

CASE VIII.—The urine before operation was negative; the urine fourteen hours after operation was negative; the urine the second day after operation was negative; the urine the third day after operation was negative; the urine the fourth day after operation was negative; the urine the fifth day after operation gave a positive reaction; and the urine the sixth day after operation gave a positive reaction. On the sixth day after operation an attempt was made to open the pancreatic duct at the point of ligation. This was unsuccessful on account of the induration about the duct. The first day following this procedure the urine was lost; on the second and third days the reaction was nega-

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tive; on the third day typical crystals were found; and on the following day the animal died. The urine collected on this day up to the time of death gave a negative reaction. We are not prepared at this time to explain this phenomena.

From the anatomical lesions produced by these experiments we inferred that a mechanical obstruction of the duct would seem to be productive of a positive Cammidge reaction. This led us in Case VIII to attempt the removal of the obstruction caused by the ligature. Our purpose was to see if the reaction would disappear after the pancreatic duct again became patulous. As the reaction did not appear until the fifth day, and as by this time the induration was very marked, the attempt to pass a probe through the duct from its intestinal orifice was unsuccessful.

While not regarding the results of our experiments as conclusive, we believe them, nevertheless, to be very suggestive. Further research has been undertaken to determine the factors concerned in the production of the pancreatic reaction. The results of these studies will be published in our final report.

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CIRCUMSCRIBED SEROUS SPINAL MENINGITIS.[1]

A LITTLE-RECOGNIZED CONDITION AMENABLE TO SURGICAL TREATMENT.

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(From the Department of Neurology in the University of Pennsylvania.)

A COLLECTION of clear fluid in a cyst of the spinal pia-arachnoid is a condition little known in America, as only one case occurring in this country is on record (Spiller, Musser, and Martin, 1903), and only a few are to be found in the German literature. So far the French and English journals contain no examples. A recent excellent *resume* of the subject by S. Adler makes a complete critical digest by me unnecessary, and yet, because of the little attention paid to the condition, it has seemed desirable to bring the subject before the College.

Almost nothing is known as to the origin of apparently idiopathic collections of fluid within the pia-arachnoid. The fluid may be from the bloodvessels of the pia, as has been suggested, and the mesh-like structure of the leptomeninges may predispose to adhesions, but why these cysts occur in any given case, especially where there are no evidences of inflammation, we do not know. The symptoms are those of circumscribed compression of the spinal cord, and the condition cannot be distinguished clinically from tumor of the cord. It is of great importance, as operation is easy and cure seems to be permanent in typical cases.

In the report of the case of Mendel and Adler, in which an oper-

[1] Read December 2, 1908.

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ation was performed by the latter, it is stated that the much swollen arachnoid protruded through the dura at the operation and appeared violet blue in color, apparently exactly as in the case we reported. The arachnoid was punctured, and about a teaspoonful and a half of clear fluid escaped in a strong stream and was lost in the bandages. The intra-arachnoidal cavity in which the fluid had been contained was found closed at all parts by adhesions. The improvement after this operation was very marked.

Krause and Oppenheim made their first report of this condition in 1906, but they did not refer to previous work on the subject. This was not extensive, and consisted of the reports of three cases (Schlesinger, Schwartz; and Spiller, Musser, and Martin[1]). Schlesinger, in his well-known monograph on spinal tumors, gave the history of a case in which a cyst of this character was found at necropsy. Schwarz's case (1897) was one with the symptoms of syphilitic spinal meningitis, and after death intense meningomyelitis was found, with a meningeal cyst. This seems to have been a case different from the typical examples, in that closure of a space within the pia-arachnoid might easily occur from the intense meningeal inflammation. Neither of these cases was with operation. The first case reported anywhere with operation was the case by Spiller, Musser, and Martin.

In that case a delicate pial cyst was found and evacuated by Dr. Edward Martin. The operation was on June 30, 1902. The patient had suffered intensely. She has been reëxamined by Dr. Musser and me within the past few weeks, and she has now no symptoms of her former trouble except a slight pain in one buttock when she presses over the scar made by the operation. She has gained much in weight and is in excellent health.

This is not only the first case with operation, but is the only one on record in which recovery has persisted after operation so long as six years and five months.

Krause, in operating on 22 cases of spinal compression, expecting to find tumor, found circumscribed serous meningitis in 6, although it is not certain that this was the only lesion. Adler remarks that the condition is not a scientific curiosity, as some may think,

[1] University of Pennsylvania Medical Bulletin, March and April, 1903.

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but is of practical significance. He refers to a case seen by Stroebe. A very similar condition on the under surface of one cerebellar lobe has been described by Placzek and Krause.

The associated lesions that have been found are: Necrotic ostitis of the vertebræ with pachymeningitis (Krause); scar-like adhesions between the dura and pia, probably with spinal gliosis (Krause); bony projection on the inner surface of a vertebra (Krause); and meningomyelitis (Schlesinger, Schwarz).

As Adler remarks, no cause was found in the case reported by him with Mendel, nor in one case of Krause, nor in the case of Bruns, nor in our case. Adler is inclined to believe that in the case reported by himself and Mendel tuberculosis may have been present, as the patient had had pulmonary tuberculosis; and he hesitates on the basis of four cases to speak of an idiopathic form of circumscribed serous meningitis. He suggests the possibility of spinal puncture, and refers to the fact that after operation the fluid does not seem to collect again. Bruns, stimulated by Mendel and Adler's[1] paper, publishes his case more in detail, and reports almost complete recovery two years after the operation.

A case reported by W. C. Krauss hardly belongs to this group of cases, as the cyst was not confined to the pia-arachnoid. The diagnosis of spinal tumor was made, but an extradural cyst in eroded vertebræ was found. Dr. Roswell Park states, however: "It seemed evident that there was a cavity with eroded walls connecting with the spinal canal, containing mostly fluid, with a little semisolid tissue material."

It is questionable whether the spinal cyst described by Schmidt should be included under this type. It was intradural, but not a cyst of the pia. Its walls had the consistency of the dura.

Although the literature on circumscribed serous spinal meningitis, as shown by this *resume*, is meagre, it is sufficient to stimulate us to pay attention to the condition and to encourage us in the belief that certain cases with the symptoms of spinal tumor may be more amenable to surgical intervention than are many tumors.

[1] Berl. klin. Woch., August 31, 1908. The literature is given in this paper; Bruns, ibid., September 28, 1908; Krauss, Brain, 1907, xxx, 533; Schmidt, Deut. Zeitsch. f. Nervenheilkunde, 1904, xxvi, 318.

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A RÖNTGENOGRAPHIC STUDY OF PERISTALSIS—THE RELATION OF WAVE FORM TO FUNCTIONAL ACTIVITY.[1]

By CHARLES LESTER LEONARD, A.M., M.D.

Recent studies upon the living subject show that the normal stomach varies widely in shape and anatomical relations in different individuals. These normal variations differ entirely from the classical description of the anatomists. In fact, the physiological variations are so great that no type has been agreed upon as normal. The conclusions to be drawn is that the shape varies with the characteristics of the individual, and that the stomach is normal which fulfils its functions perfectly.

The stomach is a freely movable distensible organ suspended at its cardiac and pyloric extremities. It varies in its size and shape with the amount of food ingested and the support given it by the other viscera under an intra-abdominal pressure exerted by the abdominal parietes. Its lower border and walls are freely distensible, while its lesser curvature is more rigid. It, however, changes its length and position in a remarkable manner even under normal conditions. It extends in length with an increased load, so that the pyloric end is often pushed far over to the right and curves back upon itself to the pylorus.

Deficient functional power may be present in stomachs that conform in shape to those having perfect function. Gastrectases, gastroptoses, and pyloroptoses have been described as pathological conditions. The significance of form can only be determined by

[1] Read December 2, 1908.

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estimating the functional efficiency in the individual case. This functional efficiency can only be determined by the ability of the stomach to empty itself in a given time.

Exclusive of newgrowths, ulcerations, scar-tissue contractions and ptoses, gastrectases are the pathological conditions that most frequently interfere with functional efficiency. They may arise from two diametrically opposed states. They are on the one hand the passive dilatation due to muscular atony, overdistention, and lack of intra-abdominal support, and on the other hand dilatation with hypertrophy of the muscles caused by organic or spasmodic stricture of the pylorus. They are, therefore, sthenic and asthenic forms of gastrectasis in which functional efficiency is defective, as shown by the residue of an ingested meal after the normal period in which the stomach should have emptied itself.

The presence of this abnormal residue is determinable by employing the bismuth meal and the Röntgenographic method of examination. This, however, determines only a lack of motor efficiency, and does not differentiate between the sthenic and asthenic forms.

Is it possible for the Röntgen method to differentiate in any way between these two forms of motor insufficiency?

The study of the stomach by the Röntgen method of examination has shown, in addition to the determination of motor insufficiency, variations in the size, shape, and position of the stomach. It has in addition determined the presence of newgrowths and scar-tissue contractions invading its lumen. The results of these studies upon the normal position and shape of the stomach and their pathological variations have been discussed at length by various authors, and will not be considered in this paper. This study is confined to variations in the form of peristaltic waves in their relation to normal and pathological conditions, and their relation as a possible means of differentiating between the sthenic and asthenic types of gastrectasis.

Recent advances in apparatus and technique have made it possible to make instantaneous Röntgenograms of the stomach and intestines, which show some of the various forms of peristaltic

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waves under different normal and pathological conditions of the stomach. Although they are not as yet so extensive or complete as to justify absolute conclusions, certain deductions can be drawn with reference to the relation of peristaltic wave form to functional efficiency, which will aid in differentiating the sthenic from the asthenic pathological conditions of the stomach.

These studies show that there is a constant relation between the form and amplitude of the peristaltic wave and the amount of work to be accomplished, that the amplitude of the wave varies with the character of the food ingested, the position of the patient, and the amount of the gastric contents. They have shown that the amplitude varies in pathological conditions. The variations in sthenic and asthenic forms of gastrectasis are so distinct that apparently a differentiation can be made between gastrectasis due to a weakened musculature and that due to pyloric stenosis, whether organic or spasmodic. In the asthenic type the waves of peristalsis are practically absent when the patient is in an erect position, and of but slight amplitude when the patient is prone or recumbent. In the sthenic type the wave is of greater volume and less frequent, but particularly strong when the patient is prone. The therapeutic value of rest after eating for all cases of motor insufficiency is thus demonstrated since the waves are increased in strength.

The study of the peristaltic waves in the normal stomach under varying conditions, in the kind and amount of food ingested, shows that in the human subject the conditions are practically identical with those determined by Cannon in his classical studies on animals with the bismuth meal and phoroscope.

The difference between solids and liquids is seen not only in the stomach, but also in the esophagus. Boli of bismuth in bread and milk passing separately along the esophagus, while bismuth in kumiss shows the entire shadow.

The peristaltic waves vary in volume with the amount of food ingested, but are alike in type for the same kind of food, the shape of the waves differing when fluids or semisolids are employed.

When bismuth is taken in a glass of water the water is rapidly absorbed and the stomach is shown in its resting or nearly empty

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state with a fine saw-tooth form of peristaltic wave. As the load increases the amplitude of the wave increases while its sinuous type persists. With semisolid contents, as bread and milk or rice-pudding, the waves become less frequent with a greater amplitude, which shows that more work has to be done.

These characteristics are most prominent when the patient is prone, but retain their individuality of type when the erect position is assumed. In gastrectasis of the asthenic type all waves are practically absent when the patient is in the erect position. In the prone position they present the normal type, but are less frequent, while their decrease in amplitude shows the motor insufficiency. In gastrectasis due to obstruction the volume of the wave is more pronounced, although they are less frequent and are still more marked with the patient in the prone position, their increased amplitude showing that more work has to be accomplished.

The variations in the character of food ingested have not as yet been sufficiently great to determine the relative rapidity of their discharge from the stomach and passage through the intestines. They indicate, however, the possibility of further studies of great physiological interest.

One physiological fact, which had been suggested previously as possible, they have established, that is that the stomach elongates to a marked extent during expiration.

In one instance the difference in the perpendicular length of the stomach was one and one-half inches greater during expiration than in inspiration, while the base of the stomach remained at the same level. This motion may, in a measure, account for the ejaculatory expulsion of food from the pylorus, which has been observed by some operators as distinct from the flow resulting from peristaltic action.

Marked changes have also been noted in the relation of the pyloric portion of the stomach to the pylorus itself, the stomach passing to the right beyond the pylorus and curving back upon itself.

The study of intestinal peristalsis has demonstrated differences in their character and in the shapes of their contents, but

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no segmentation, as described by Cannon, in his observations on animals, has been shown. The peristaltic motion of the small intestine is so rapid that one-half-second exposures are necessary to sharply define the fecal masses in the duodenum. Their shape differs entirely from that seen in the large intestine, indicating the presence of secondary waves of less volume. They apparently decrease in rapidity as the cecum is approached.

In the large intestine the subdivision of the intestinal contents is remarkable because of its completeness. This almost complete segmentation of the fecal masses extending throughout the entire colon indicates that the process of absorption continues throughout its entire length. Various degrees of enteroptosis have been observed. That of the hepatic flexure is most common and often includes the entire ascending colon, while ptoses of the splenic flexure and descending colon are rare. These ptoses seem to have little effect upon the motor efficiency of intestinal peristalsis, since the passage of the bismuth meal is apparently not delayed in cases where they are present. They have, however, been found in cases suffering from intestinal indigestion, although their relation to this condition cannot be inferred, since they are found in other patients.

The plates to be shown illustrating these studies were made with an exposure of from one-half to one second. The meal employed has been two ounces of bismuth, either subnitrate or carbonate, in water, kumiss, bread and milk, or rice pudding. Observations were made during the act of swallowing immediately after the ingestion of a part or the whole of the meal, and at intervals of one hour, six hours, and twenty-four hours after eating.

The accompanying plates illustrate the varying phases of peristaltic motion in some of the cases upon which this paper is based. Many of the patients presented marked gastric and intestinal symptoms, but those in which no motor insufficiency was present have been considered as normal in the type of peristalsis, since they did not differ from the normal cases studied.

The first illustrations show the difference between the passage of fluids and semisolids through the esophagus.

Fig. 1 shows the entire esophagus coated by two swallows of

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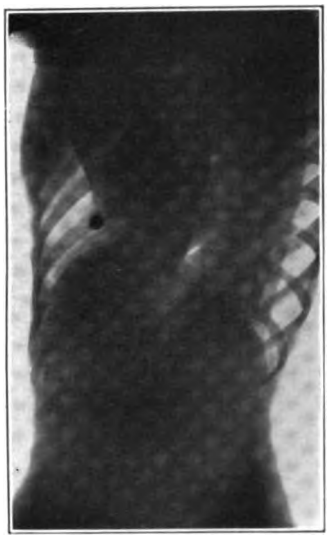

Fig. 1

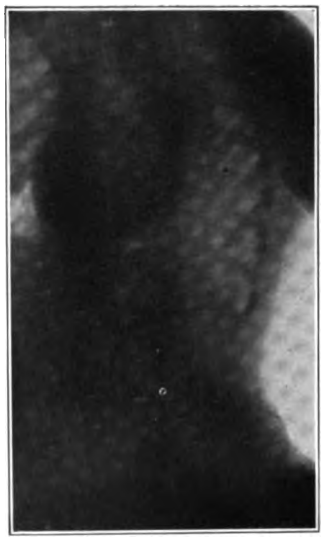

Fig. 2

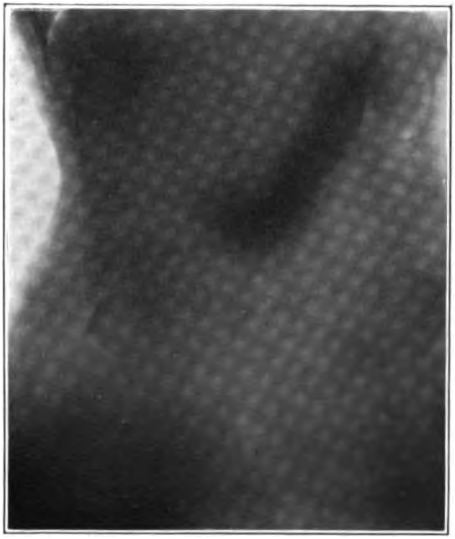

Fig. 3

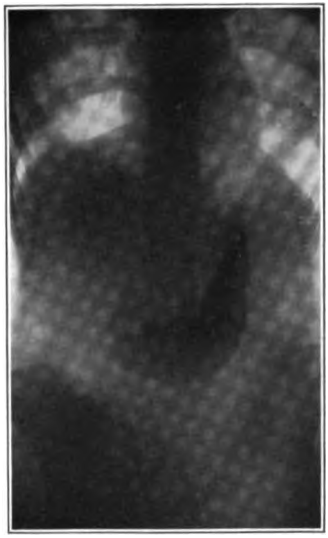

Fig. 4

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Fig. 5

Fig. 6

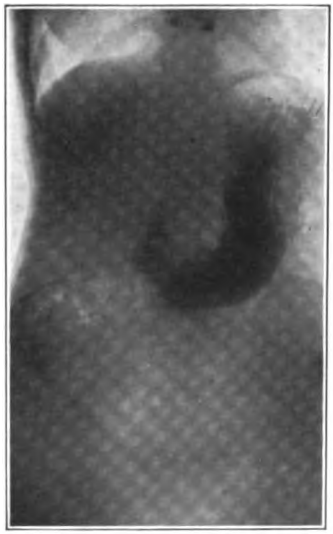

Fig. 7

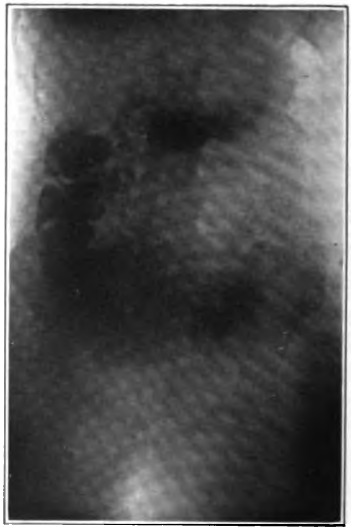

Fig. 8

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bismuth in kumiss. It was taken with the patient in the right oblique position and shows the relation of the esophagus in the mediastinum to the heart and spinal column.

Fig. 2 shows two boli of bread and milk with bismuth passing down the esophagus. The lower bolus is arrested at the diaphragmatic opening, in which a small portion is seen as a dark line projecting downward through the diaphragm.

Fig. 3 shows a normal stomach in a state of rest, the bismuth having been given in a glass of water. The stomach in this instance has the form of a distended intestine with a very fine saw-tooth type of peristaltic wave, indicating that very little motor activity is demanded.

Fig. 4. The stomach is here partially filled with four ounces of a bismuth and kumiss meal. There is a distinct "magen blasse" filling the cardia. An undulatory peristaltic wave of small volume is showing on the greater curvature of the stomach.

Fig. 5. This is the stomach of a man, aged fifty years, with two ounces of bismuth subnitrate in a half-pint of kumiss. The undulatory character of the peristaltic wave with fluid ingesta is striking. The fluid media with the bismuth can be seen rapidly passing through the pylorus, the ascending, the descending, and the reascending limbs of the duodenum. The exposure in this case was one second, and was not short enough to show the peristaltic waves of the duodenum.

Fig. 6. The bismuth meal consisted of bread and milk, and the stomach is partially filled, with the patient in the erect position. The level of the fluid in the stomach is clearly defined with the "magen blasse" or gas-filled cardia above, while below the level of the fluid is a second line which corresponds to the upper limit of the denser portion of the meal. Single undulatory waves of peristalsis of slight volume are seen along the greater curvature.

Fig. 7. In this patient the bismuth was given in eight ounces of rice-pudding. The peristaltic waves are seen to differ from those of the fluid meal, and are typical of the more solid gastric contents. The wave is seen to be deeper and single, showing on both the lesser and greater curvatures. The longitudinal rugæ are dis-

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tinctly seen filled with the bismuth meal, where the stomach has widened in its upper portion after the passage of the peristaltic wave.

Fig. 8 shows the gastric residue and intestinal contents six hours after the bismuth meal in a case of defective motility of the asthenic type. The weak peristaltic wave is seen in the stomach, although the patient was in the prone position. The entire small intestine is shown filled with the bismuth meal.

Fig. 9 shows the large intestine of the same case as Fig. 7, six hours after the ingestion of the rice pudding and bismuth.

The distinct segmentation of the feces is noted throughout the entire colon. There is a complete ptosis of the ascending colon and hepatic flexure carrying with it the transversed colon. The splenic flexure is seen in its normal place with gaseous contents, while an accumulation of gas is also seen in the cecum. This plate was taken with the patient in an erect position. In another plate taken in the prone position about the same time, there is no alteration in the position of the intestine, but the gas is absent from the cecum.

Figs. 10, 11, and 12 are from the same patient and are of particular interest, because they show motor insufficiency due to pyloric stenosis, the result of ulceration and spasm of the pylorus, which was demonstrated by a subsequent operation. In Fig. 10 we see a marked peristaltic wave in the lower third of the stomach nearing the pylorus. It involves both the lesser and greater curvatures and corresponds to what has been described as the "sphincter antrum pylori." A second commencing wave is seen between the middle and upper thirds of the stomach, while the perpendicular rugæ of the cardia and left side of the stomach are shown by the bismuth remaining in them as the stomach expands laterally. It should be particularly noted that the lungs are filled with air, shortening the sagittal length of the stomach, while its base is at the iliac crest or a little below it. When this plate is compared with Fig. 11 of the same stomach, taken immediately after the other, but with full expiration instead of full inspiration, the contrast in the length of the stomach is easily noted. The base remains at the same level,

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Fig. 9

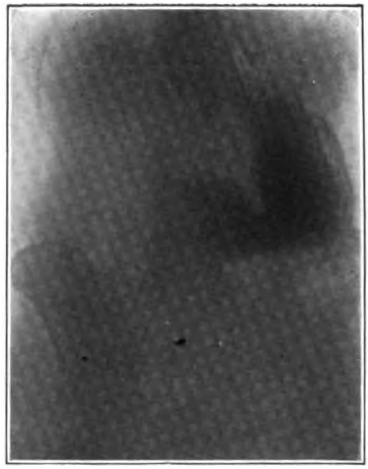

Fig. 10

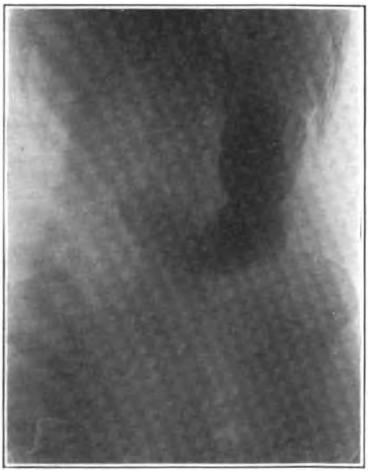

Fig. 11

Fig. 12

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only it is narrower, while the peristaltic wave, called the "sphincter antrum pylori," has disappeared and a second wave is shown in the middle third of the stomach. In addition, a very small notch on the outer border of the stomach between the upper and middle thirds indicates the commencement of another peristaltic wave.

The third plate, Fig. 12, shows the stomach and intestines of the same patient six hours after the bismuth meal. The residue of the bismuth meal is shown in the lower part of the stomach with marked peristaltic waves. Bismuth is seen in the pylorus and the descending and ascending links of the duodenum. The greater portion of the meal has, however, passed into the cecum and the ptosed ascending colon, but the transverse and descending portions of the colon are empty. This is in marked contrast to the condition of the colon in Fig. 9, where the meal has passed into the descending colon in the same length of time. In this plate the gas-filled portion of the colon can be seen, and also very distinctly the spleen and lower border of the liver. The presence of this residue in the stomach shows motor insufficiency, but the character and strength of the peristaltic wave indicates no lack of muscular power, so that some form of obstruction was diagnosticated. The subsequent operation disclosed an ulcer near the pylorus with a spasmodic stricture.

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TUMORS OF THE BREAST IN CHILDHOOD.*

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The recent occurrence of two cases of tumor of the female breast in children in our own practice has induced us to make some further study on the general subject of tumors of the breast in childhood. A search of the literature has shown an apparent absence of systematic papers on the subject, and we have thought it worth while to collect and analyze the scattered cases of these unusual affections. To the paper of Malapert and Morichau-Beauchant[2] we are indebted for the cases of angioma of the breast included in our own series. The remaining cases have been collected from the literature at large, mainly from case reports in the journals. In addition to the complete histories of our own cases, we have made brief abstracts of all the other cases which we could find and analyzed them with reference to the pathology, symptomatology, and treatment.

A large majority of tumors of the breast in childhood are benign in character. They may be conveniently divided into benign tumors of vascular origin and benign tumors other than those of vascular origin.

Vascular tumors of the breast exist as cutaneous, subcutaneous, or intraglandular formations. This subject has been thoroughly

* Read May 6, 1908.

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investigated by Malapert and Morichau-Beauchant,[2] from whose article on the subject most of the data here collected has been obtained. They have collected and analyzed all the recorded cases of angioma of the breast; among these were six, including one of their own, in children under sixteen years of age. The first class are unimportant, have the characteristics of nevi occurring elsewhere, and are apt to be seen on or near the nipple. Bryant[3] reports a case the size of a shilling, which was destroyed by the galvanic cautery.

In the subcutaneous variety the cellular tissues are invaded by small tumors which may cause atrophy of the glandular structures by pressure. They are small, nodular, circumscribed, and the overlying skin is healthy. The intramammary forms of glandular tumors are true angiomata of the breast, although it is difficult in certain cases to recognize the precise origin of the tumor. If situated near the nipple the glandular tissues may undergo atrophy, but the ducts persist because of their greater resisting power. The affection may be diffuse, as in Bajardi's[1] case, or, less often, encapsulated. Both sexes are affected with equal frequency, the growths occurring either at birth or in the earliest months of life, and they may exist for years if not treated. The symptoms vary; at times an erectile tumor may be present, which is painless, the skin normal except for the presence of a few enlarged veins. In other cases, cysts may exist due to the presence of degeneration in the tumor. In outline the tumors are usually smooth, sometimes nodular, may fluctuate if cystic, are of slow growth, and may bleed if ulceration of the skin occurs. There is a decided tendency toward involvement of the right breast, as shown in six of the eight cases reported; in one instance only (Bittner) was a bilateral tumor present.

The treatment of vascular tumors of the breast depends upon their seat and size. Small superficial tumors require no treatment, or may be destroyed by the cautery, as was done in Bryant's case. The larger tumors, situated in the breast proper, require extirpation, and in some instances are of such size that complete removal of the breast may be necessary.

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The reported cases of this type are as follows:

1. Malapert.[2] Girl, aged twelve years, developed an enlargement of right breast fifteen days after birth; and at the age of six months a decided enlargement was present. There was no pulsation in the tumor, which did not enlarge on straining and was painless. On palpation a smooth, fluctuating tumor, the size of an orange, was apparent, the skin free, the mass movable on the underlying structures. Operation: Removal of three cysts and a solid mass, the microscopic examination showing an angioma with some breast tissue remaining.

2. Bajardi.[1] Child, aged two years, tumor of right breast noted six months after birth, later attaining size of an orange. Skin blue around nipple. The tumor was slightly adherent to nipple, which was slightly retracted, and on palpation was soft and elastic. It was reducible and was made tense on straining. Operation: Excision of tumor and nipple. Microscopic examination: Angioma with atrophy of breast tissue.

3. Colzi.[5] Boy, aged ten years, had enlarged right breast since early infancy. The tumor grew slowly until the whole gland and nipple were involved. The microscopic examination of the tissues removed showed an angioma and pressure atrophy of the breast.

4. Althorp.[4] Boy, aged seven years, tumor in right breast since birth, very slow in development until the subject of trauma, when it increased rapidly. The skin became discolored; the superficial vessels enlarged. The tumor was lobulated, movable, and fluctuating. Operation: Excision of cystic tumor containing thirty ounces of fluid. Microscopically the mass consisted of angiomatous and muscular tissues.

5. Williams.[6] Boy, aged seven years, developed a tumor in right breast, outside and below nipple, which was not involved. The growth was of five months' duration, about the size of a half-crown, and firmly adherent to the breast. Diagnosis: Degenerated angioma. Operation: Removal of breast.

6. Bryant.[3] Girl, aged fifteen weeks; tumor involved whole breast and skin, nipple retracted. The tumor measured two inches in diameter, was spongy and prominent, covered by large veins. The whole swelling could be reduced by pressure. No treatment was instituted.

BENIGN TUMORS OTHER THAN THOSE OF VASCULAR ORIGIN.

As in the adult breast, we encounter a variety of benign tumors of this type in the mammary gland in children. In describing these types they may be grouped clinically as benign, as the symptoms they produce are fairly constant. In most of the cases the tumors consist of an hypertrophy of both fibrous and epithelial

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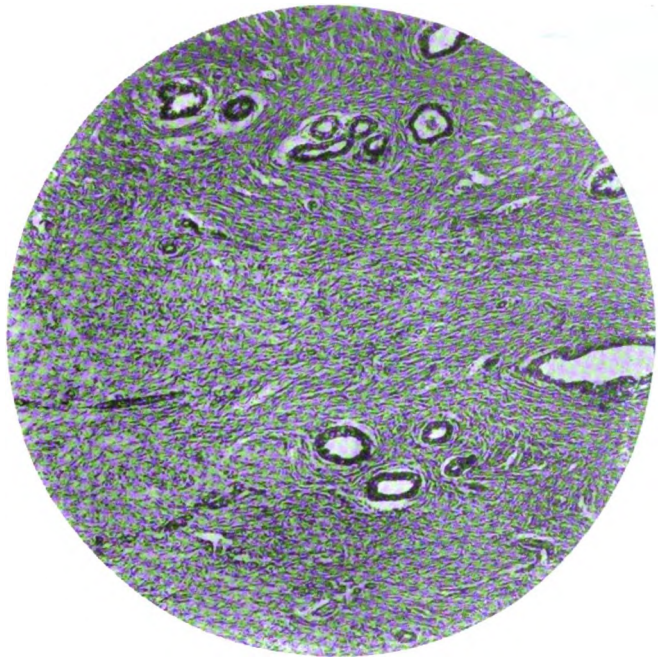

FIG. 1.—Case I. Adenofibroma of the breast in a girl aged ten years.

FIG. 3.—Histological structure of the tumor in Case II. It is made up for the most part of elongated spindle-shaped cells arranged in whorls.

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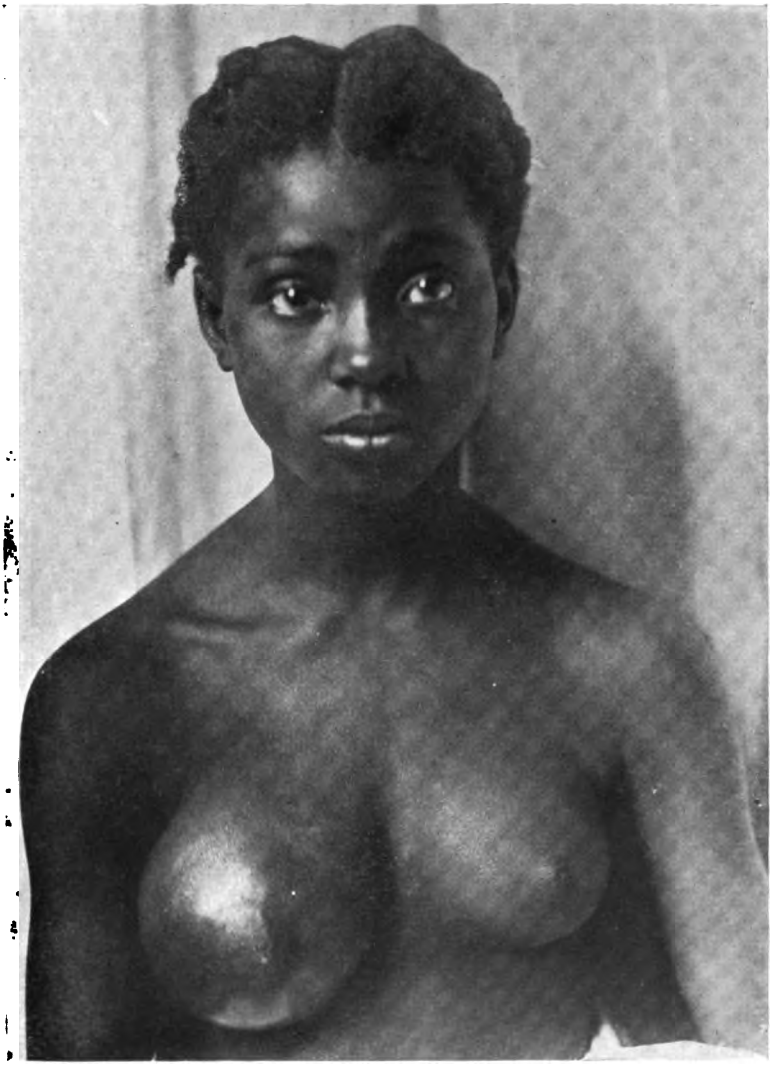

FIG. 2. —Case II. Diffuse fibroma of the breast in a girl aged eleven years. The general enlargement of the gland, tense and shining integument, and dilated subcutaneous veins simulated a sarcoma.

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elements, giving the usual appearance of fibro-epithelial growths as seen in the adult, and can be classified, therefore, as fibro-adenomata. A few instances of cystic disease of the breast and of lipoma in children are on record.

The two cases which follow were observed by us, and are reported for the first time:

CASE I.—F. W., female, aged ten years, was first seen in January, 1907, in the Surgical Dispensary of the University Hospital. The child was poorly nourished and slightly anemic. She complained of pain and tenderness in the left breast, particularly in the region of the nipple. There had been no traumatism suggestive of a cause of the pain. On examination the breasts were found to be in an undeveloped state, although the left one was slightly larger. Palpation caused some pain, and revealed a slight thickening of the tissue beneath the skin; local measures were instituted and the mother advised to have the child return if she noticed any subsequent enlargement of the breast. The condition remained unchanged for ten months, when there was a decided increase in the size of the breast, which became more painful, especially if handled. When examined one year after the first visit and two months after this decided enlargement, a small mass the size of an almond could be readily palpated. It presented the usual symptoms of a small adeno-fibromatous growth, being freely movable, and the overlying skin not affected. Enlarged axillary nodes or dilated veins were not present. Because the tumor was in intimate relationship with the nipple, the possible danger of atrophy of the breast following removal of the growth was considered and explained, and operation advised in view of the possible malignancy due to the sudden increase in size. The operation was performed by Dr. John Speese. The rudimentary gland was exposed by an incision one and one-half inches in length at its base, and the tumor removed. It was encapsulated, slightly adherent to the portion of the gland beneath the nipple, so that in its removal it was impossible to avoid some injury to the ducts; it was easily stripped from the pectoral fascia. The incision was closed by a few interrupted sutures, and the wound healed rapidly with practically no scar. Microscopically, the tumor consisted of a fibro-epithelial formation showng a typical adenofibromatous growth (Fig. 1). The glandular part consisted of a few acini, the greater portion being derived from the ducts, which were lined with cuboidal epithelium.

CASE II.—Addie P., colored, aged eleven years. Admitted to the Children's Hospital, April 23, 1907. Service of Dr. J. H. Jopson. An only child. Both parents living and well. No history of specific or malignant disease. No illness except measles and pertussis. Has never menstruated. Enlargement of right breast, stated to have been noted first by the child some eight weeks

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before. Mother has observed it for four weeks, during which time it has rapidly increased in size. Occasional sharp pains in breast for several weeks. No other symptoms complained of.

Physical Examination. Patient is well developed for her age. General nutrition good. Examination of heart and lungs negative. Abdominal examination negative. The right breast is enlarged, globular in shape, nearly three times the size of left. It is very hard. The skin is tense and shiny. Numerous large subcutaneous veins radiating from nipple. The nipple is flattened and partially retracted. No special tenderness. Breast is very movable on underlying tissues. One or two small glands are palpable in the axilla (Fig. 2). Diagnosis: Sarcoma.

Operation. April 26, 1907, amputation of breast. Elliptical incision, greater part of skin covering breast sacrificed with it. Great vascularity of subcutaneous tissue. Pectoral fascia was cleaned from muscle, which was not removed. Thorough dissection of the axilla from below. Several enlarged glands were found. Their appearance suggestive of sarcomatous infiltration. Undermining of skin edges was necessary to closure of wound, which was accomplished except for a central area about the size of a silver dollar. Pri, mary healing of wound. Patient discharged from hospital June 13, 1908.

Laboratory Report. The tumor when received measured about 6½ x 4½ x 3½ inches. The overlying skin, including the nipple, was intact and freely movable on the tumor. The nipple showed marked retraction. The tumor mass of an irregularly moulded shape, its consistency firm throughout and of marked density. Section of the tumor showed it to be composed entirely of pale-whitish, glistening tissue, poorly supplied with blood. On section the tissue was arranged in rounded masses composed of whorls of glistening white striations; this appearance was continuous throughout all portions of the tumor.

Histological Examination. Blocks of tissue for examination were taken from various portions of the tumor mass, those from the surface showing a distinct fibrous capsule consisting, for the most part, of typical old connective-tissue fibers. Directly beneath this capsule the tumor was made up of elongated spindle-shaped cells closely packed together and arranged in whorls (Fig. 3). These cells were of the fibrous-tissue type and characteristic in arrangement and number of the cells found in rapidly growing tumors composed of fibrous tissue. Numerous ducts were seen throughout the tumor, which were lined by cells of a cylindrical type, arranged in some places in one layer and in others in several layers. Some of the ducts showed an enlarged lumen resembling cystic formation, and in such localities the epithelial lining was flattened and atrophic from pressure. In no place had the epithelium broken through the basement membrane or infiltrated the surrounding tissues. The breast tissue had practically disappeared except for the presence of these ducts scattered through the tumor; and its place and bulk were represented by the new-formed fibrous tissue.

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Pathological Diagnosis. Diffuse fibroma. Histological examination of several small lymphatic glands showed moderate congestion and no evidence of tumor metastasis.

We have collected from the literature thirteen cases of this type, abstracts of which follow:

Weinokouroff.[9] Girl, aged thirteen and one-half years. Left breast noticed to be enlarged fifteen months before, but not painful. Growth stationary for ten months, when it rapidly increased until three times the size of opposite gland; no pain. Tumor occupied entire left breast, hard, size of two fists, movable. Removal of breast. Cured. Diagnosis: Adenofibroma.

Patterson.[11] Girl, aged thirteen years, tumor developed seven months after injury; slow growth, no pain or tenderness, not adherent, almond sized, lobulated. Tumor removed. Diagnosis: Adenofibroma.

Patterson.[11] Girl, aged twelve years, noted a lump in right breast three months ago; painless. Apparently disappeared, but reappeared in one month, causing pain, and gradually growing until it was the size and shape of a fig. Growth situated below nipple and not adherent, but tender and sensitive. Operation: Removal of tumor. Diagnosis: Adenofibroma.

Barton.[17] Girl, aged twelve years, swelling of left breast, when first noticed was size of pigeons' egg, and painless. In two months' time was four times size of opposite breast, covered by dilated veins, lobulated, freely movable, nipple and glands normal. Operation: Removal of breast. Diagnosis: Adenofibroma.

Cartledge.[12] Girl, aged sixteen years, right breast seat of a tumor, situated midway between nipple and axilla. Rapid in growth, painful, superficial veins enlarged. Tumor movable, size of goose egg, not adherent to gland. Operation: Entire breast removed, probably fibroma.

Hopkins.[10] Girl, aged eleven years, four years previously had tumor, size of chestnut, removed from right breast. Six months after operation left breast enlarged; later very rapid growth and enlargement of axillary nodes. Never menstruated. Entire breast and four nodes removed. Diagnosis: Fibroma. First tumor probably same type.

Le Double.[13] Girl, aged fifteen years, rapid increase in size of both breasts following cessation of menstruation. Nipple flattened, overlying skin tense and containing large veins, pain of lancinating character, discomfort from weight of breasts. Diagnosis: Hypertrophy. Under palliative treatment there was a diminution of left breast, right one increased in size and finally was amputated, weight being 1985 gm. Microscopical examination: Fibro-adenoma, preponderance of fibrous tissue.

Gueniot.[7] Observed a case in a girl aged fourteen years; breast three times its normal volume; lancinating pains and enlarged nodes of the axilla were palpable. Diagnosis not given.

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Haynes.[9] Girl, aged thirteen years, developed an enlargement of both breasts about nine months before. There was slight pain on pressure, which symptom soon disappeared. Five months after the enlargement began the left breast grew very rapidly. An examination showed that the breast extended to the ilium when the patient was standing. The nipple was flattened and the region about excoriated. The breast was freely movable on the deeper structures, but was not painful and only inconvenient by reason of its weight and size. The axillary glands were not enlarged. The breast when amputated weighed exactly eight pounds, and was reported as a fibro-adenoma undergoing mucoid degeneration.

Parsons.[10] Child, aged three years, enlargement of left breast noted eighteen months previously, slow growth, no pain or discharge from nipple. Mass was size of an egg, cystic in character, adherent to skin, nipple normal. No tenderness or enlarged nodes. Removal of tumor and part of pectoral fascia. Diagnosis: Cyst, containing clear fluid within and a smooth wall.

Spencer.[15] Boy, aged four years, lump in right breast since birth, round, tense, size of pea, painless. Rapid increase in size during the last three weeks, when it became size of egg, skin normal, no discharge from nipple, no nodes palpable. Exploratory puncture gave bloody fluid. Breast amputated. Was seat of multiple cysts and a solid growth, size of hazel nut. Probably cystic fibroma.

Atkins.[14] Records an enormous lipoma, twenty-five pounds, in a girl aged sixteen years. At the age of four years parents noticed a slight enlargement of left breast, which increased slowly and without pain until she was twelve years old. Then tumor began to enlarge very rapidly. Right breast was normal. Catamenia have not appeared. Growth removed. Operation followed by cure. Diagnosis: Lipoma.

Bryant.[13] Male, aged ten months. Hard tumor size of walnut situated above and to outer side of left nipple, moving freely upon the deeper parts, skin adherent but not discolored. Four months before began to grow rapidly. Composed of fatty and fibrous tissue.

We have carefully analyzed these 21 cases of benign tumors of the breast in so far as the data, often scanty in the original reports, would permit. We have made the usual age limit of sixteen years. In only one of the cases in the older girls was menstruation mentioned as having occurred, and in several others it was stated that it had not occurred.

First, with reference to the type of tumor and its relative frequency: 11 of the 21 cases can be classified as fibromata or fibro-adenomata. The dividing line between these two types of tumor,

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if it exists at all, is a very indefinite one, and we are inclined to follow the classification of Ribbert as adopted by Warren, Rodman, and other writers on this subject, and consider them as fibro-adenomata. They constitute the most numerous of any type of tumor of the breast occurring in childhood, and their proportion to the total number of benign breast tumors is higher in children than in adults. There were 6 angiomata, 1 fibrolipoma, 1 lipoma, and 1 simple cyst; in one case the diagnosis was not given. Fourteen of the cases were in females, 5 in males, and in 2 the sex was not stated; these were probably females. In 3 of the 6 cases of angioma the tumor was in boys. In this, as in other respects, angiomata are practically in a class by themselves as regards their occurrence, pathology, and symptomatology.

Age. Most of the angiomata were noted at birth or soon afterward; the children, however, were often half-grown before being brought for surgical attention. The oldest was aged twelve years and the youngest fifteen weeks. Of the other tumors, with three exceptions, the cases were ten years old and upward. These 3 cases were aged ten months, and three and four years respectively; the other cases ranged between ten and sixteen years; 1 was aged ten, 2 eleven, 2 twelve, 3 thirteen, 1 fourteen, 1 fifteen, and 2 sixteen years.

Duration. The duration ranged from two months to twelve years. In some cases the growths were distinctly rapid from the start, but not infrequently they were of gradual development or showed a rapid increase after a long stationary period.

As already stated, the angiomata were commonly congenital or appeared in infancy.

Causation. As to causation, in one case there was a history of traumatism before the appearance of the tumor, and in another case, an angioma, there was rapid increase in the size of a congenital tumor following injury.

Breast Involved. In 9 cases the right breast was the seat of tumor, in 7 the left breast was affected, in 2 both breasts, and in 3 the location was not stated.

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Location of the Tumor in the Breast. But little data was furnished in this respect. The growth was stated to be below the nipple in 2 cases, and twice in the outer quadrant.

In some cases the entire breast was involved, while in others the tumor was of a localized nature, and it was difficult from many of the case reports to state to which of these two classes the case should be assigned. In at least 9 cases practically the entire breast was involved.

Size. The largest tumor (Atkins[14]) was a lipoma which weighed twenty-five pounds. In Le Double's[13] case the tumor weighed between four and five pounds (a fibro-adenoma), and in Haynes[9] case (a fibro-adenoma undergoing mucoid degeneration) it weighed eight pounds. Several times the tumor was said to be three or four times the size of the opposite breast. The smallest growths were the size of a walnut, almond, or egg, and were seen to be of a localized nature.

In practically every instance the tumor or, if the whole breast were involved, the breast was said to be movable. In two of these cases the skin was not adherent.

Nipple. The nipple was retracted in several cases, flattened in two, and said to be normal in one. The skin is usually normal except for the presence of dilated veins, which are not uncommon. Dilated veins were described as being present in 5 cases, 2 of which were angiomata. In our own case, in addition to this enlargement of the veins, the skin was tense and shining; in 1 case of angioma it was said to be discolored.

Pain and Tenderness. In 6 cases pain or tenderness or both were mentioned as being present, sometimes sharp and lancinating. In 4 cases pain was said to be absent, and in the remaining instances it was not mentioned.

Lymph Nodes. These were enlarged in 3 cases, not enlarged in 5 cases, and not mentioned in the remainder.

Operation. In 11 cases the entire breast was removed, and in 2 the axilla was dissected free of enlarged lymph nodes. In 8 cases the tumor was excised, once including the nipple. In one case no treatment was instituted, and in one it was not mentioned.

Result. So far as known all the cases operated upon were cured.

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MALIGNANT TUMORS. CARCINOMA AND SARCOMA.

The number of malignant tumors of the breast which have been observed in childhood is small. This is what might be expected in the case of carcinoma, the rarity of which is well known in childhood. It is doubtful if any well authenticated case of carcinoma of the breast under sixteen years of age has been reported, and Gross states that it does not occur under the age of twenty years.

P. W. Phillip,[28] in his article "Ueber Krebsbildungen in Kindersalter," collects 390 cases of cancer reported as occuring in childhood. Of this number, only 87 (22.3 per cent.) withstood critical examination as to their nature, the remainder being doubtful cases. This is sufficient to show the rarity of cancers in childhood. Phillip did not find a case of mammary carcinoma under fifteen years, and only 1 case of cancer of the uterus, while 26 cases of ovarian cancer were observed. There seems to be a relative predisposition of the ovaries to malignant disease in childhood, furnishing 20 per cent. of all cases of cancer, while in adult life they furnish but 5 per cent. at the highest estimate. We have found but three cancers of the breast in children, and all of these cases are doubtful in nature, and reported many years ago. They are as follows:

1. Lyford.[19] A case in a girl, aged eight years. (Mentioned in John Birkett's article, "Diseases of the Breast," in Holmes' *System of Surgery*, vol. iii.)

2. B. B. Cooper.[20] Lectures on Surgery, 1851, reports a case which he thought to be cancer in a girl, aged thirteen years. The tumor was rapid in its development, painless, and accompanied by cachexia and metastasis to the thoracic and abdominal viscera. (This may have been and probably was a case of sarcoma.)

3. Birkett.[21] Mentions a specimen in the Museum of St. Bartholomew's Hospital, removed from a girl, aged sixteen years.

SARCOMA.

This also is a rare disease of the breast in children. While a number of references are contained in the literature, very meagre descriptions of the histological structure of the supposed sarcomata

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are furnished. Gross[29] states that 1.66 per cent. of cases of sarcoma of the breast occur during the developmental stage, that is, before the sixteenth year. Karewski[31] collected 156 cases of sarcoma of the breast, of which 1 occurred before the tenth year and 14 before the eighteenth year of life. There is no doubt that sarcoma is a much rarer tumor of the breast than was supposed by the older writers, a fact recently emphasized by Rodman[30] in his work on *Diseases of the Breast.* Instead of 5 to 9 per cent. of mammary tumors, he estimates its frequency from an analysis of 5000 cases at 2.78 per cent. The relative liability of the female breast to sarcoma is below that of the body in general, and while, heretofore, it has been generally assumed and stated that it was a disease of young women, it is a fact, as Rodman points out, that one-half of the cases occur between the ages of forty and fifty years. Two explanations may be furnished to reconcile the disparity between the figures of the older writers, as Gross, and those of the present day. The commonly accepted one is that a more accurate histological diagnosis is now insisted upon, and that the microscope is relied upon more than the clinical picture. Another explanation which has occurred to us is, that the great increase in recent years in the number of cases of cancer of the breast, as of cases of cancers of all kinds, has not been accompanied by a relative increase in the number of cases of sarcoma of the breast.

Types of Sarcoma Found in Childhood. Generally speaking, spindle-cell tumors develop at an earlier age than do round-cell tumors, while cystic sarcomas are observed more frequently in youth. It may be difficult to diagnosticate between a rapidly growing fibroma and a spindle-cell sarcoma. Thus in our second case, diagnosticated before operation as sarcoma, several experienced pathologists made a diagnosis of sarcoma, from some of the microscopic preparations, which apparently confirmed the clinical diagnosis. The liability to error before operation is not inconsiderable, for our second case presented the clinical picture of a rapidly growing sarcoma, the diagnosis being based upon the rapid growth of the tumor, its general appearance and the presence of the distended veins in the overlying skin, an appearance which is common to

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sarcomata in general. A review of the cases here collected will show, however, that this latter symptom is described as present in a number of cases of non-malignant tumors of the breast in childhood, both of angiomatous and fibro-epithelial origin. The following are the references to sarcoma of the breast. Scant details are furnished in the original reports.

1. Sheild.[22] A rapidly growing spindle-cell sarcoma in a girl aged fourteen years.

2. Sheild.[23] A myxosarcoma removed from the breast of an infant aged six months.

3. Chambers.[27] A four months' old child presented a small round-cell sarcoma of the breast the size of an egg. Duration, since five weeks. Removed. No recurrence at the age of five months.

4. Billroth.[25] A child, aged nine months. Sarcoma of breast. Removal.

5. Hansey.[26] A case seen at an early age.

6. Rodman.[24] A girl, aged eleven years, gave a history of traumatism one year before development of the tumor. Pain in breast was followed by an increase in size, the growth being fairly hard. Removal was advised on account of increased pain and the presence of enlarged veins in the overlying skin, which the author believed indicated the tumor to be of sarcomatous type. Breast was removed. No enlarged glands were found in the axilla. Microscopic examination not made, specimen being lost.

The diagnosis was based on what may have been misleading clinical symptoms. The presence of the enlarged veins is not conclusive evidence of malignancy as shown in our own case and in several of the vascular and fibro-epithelial tumors which have been collected in this series.

CONCLUSIONS.

Tumors of the Breast. While rare in childhood, these occur in both sexes and at all ages.

The benign tumors are more frequently encountered in the mammary gland in early life than the malignant tumors. The fibro-epithelial growths are the most numerous group of the benign tumors, and next to these in point of frequency come the angiomata.

Sarcoma may occur in children in the mammary gland, but it is a rare tumor. The breast enjoys almost complete immunity to carcinoma before the age of puberty.

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Girls are affected more frequently than boys, but the disparity in numbers is immensely less than in adults.

The angiomata are commonly congenital, or first appear in infancy. The fibro-adenomata tend to develop more frequently as the child approaches puberty.

Some of the smaller benign tumors occasion no inconvenience. Others are associated with symptoms, pain, tenderness, and inconvenience or discomfort from excessive weight or size. Sarcomata present the symptoms common to that type of tumor.

Operation is usually indicated in the benign and always in the malignant varieties. In small benign tumors, or those involving only limited areas, conservative plastic operations with preservation of breast and nipple are indicated. In a goodly number, however, the breast must be sacrificed. The axilla should be cleaned if it contains enlarged glands. The results of operation are good.

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THE APPEARANCE OF PELLAGRA IN THE UNITED STATES.[1]

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It is quite probable that pellagra existed for many years in Europe before its recognition as a distinct disease. It was long considered an unusual manifestation of some other disease, as scurvy or leprosy. The first indication of the disease in literature was made by Ramazzini, who called it *mal del padrone*. In 1730 Gaspard Casal, a physician of the Asturias, described pellagra under the name *lepra asturiensis*. This name is accounted for by the fact that leprosy was at that time prevalent in the Asturias, and Casal considered pellagra a manifestation of leprosy. Another author accredits the name *lepra scorbutica* to Casal. Soon after this it was noted in Galatia, and somewhat later in Castile, Rome, and Aragon. Among the people it was variously designated as *mal de la rose, mal del sole*, or *mal de la misera*. About 1750 the Northern provinces of Italy, chiefly in the neighborhood of the Lago Maggiore, became the chief seat of the disease, and seemed to furnish a much more favorable field for its extension, and while the disease was still present in the lower Spanish provinces, it has never made as much headway there as in Italy.

Frapalli was the first to apply the name pellagra (Ital. *pelle* and *agra*, rough skin). This was about 1771. Frapalli claimed that the disease pellarella, which had been known for many years, was identical with pellagra, and that it was present before the introduction of Indian corn from America. Contemporaneously with

[1] Read by invitation, November 4, 1908.

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Frapalli the disease was described by Odoardi in Venetia, and called by him *scorbutus alpinus*. Somewhat later, Puyati did important work in showing that all these descriptions and names were for one and the same disease—pellagra.

By 1784 the disease had reached such proportions and the people had become so aroused that the Joseph II Pellagra Asylum was established in Legnano under royal authority and the elder Strombio placed in charge of it. Strombio's work was of the greatest importance, and marked a decided advance in the study of the disease. By the close of the eighteenth century it had extended into Liguria and Piedmont. In 1818 Hameau, the elder, described it under the name *mal de la teste*, occurring in the neighborhood of Teste, and it was at this time that the people bestowed the appropriate name *mal de la misera*. It was also known as *dartres malignes, mal de Saint Rose, mal de la Saint Mari*, and numerous other names of similar character, after the holy orders to which the sufferers went for relief.

In 1826 Marchand recorded the appearance of pellagra in the southern provinces of France, especially in the rural districts, in the Gironde, in the Pyrenees, and in the Haut-Garonne and the Aude.

In recent years in these latter districts it has been greatly reduced, presumably because the corn culture has largely given place to the cultivation of other grain for which the soil is better suited.

Roumania, which is now a stronghold for pellagra, had no recorded case until 1810. This date is associated with the introduction of Indian corn from Italy in coastwise vessels. In 1836 Varnow described it in his inaugural dissertation, and in 1858 Julius Theodori did likewise. The people knew the disease by the name *Buba Tranjilar*.

In more recent years Bouchard described the disease in Mexico. It has occurred in Brazil, Argentine Republic, Uruguay, and also in Africa. There have been sporadic cases reported from the Tyrol, Servia, Bulgaria, Greece, Asia Minor, and one case from England. In the 60's, Gray, of New York, and De Wolfe, of Nova Scotia, reported sporadic cases, but the correctness of the diagnosis was questioned.

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Pellagra has without doubt existed in this country for many years, but the appearance must necessarily have been sporadic, for no death-dealing pestilence such as the acute pellagra now so prevalent in the Southern States could have long been overlooked.

In 1902 Harris, of Georgia, reported a case simulating pellagra, in which he found abundant hook-worms. From the description the case must have been well-defined pellagra.

In 1883 Sherwell reported a case in New York. The patient was a Genoese sailor. We are indebted to G. H. Searcy, of Alabama, for the first positive report. He recorded an epidemic of 88 cases occurring in the State Hospital for Colored Insane at Mt. Vernon. Of this number, 57 died. In 1907 J. T. Searcy reported 9 cases from the Bryce Hospital, in Alabama. T. C. Merrill, in 1907, reported a sporadic case from Texas. Recently, before the Medical Section of the American Medical Association, R. H. Bellamy reported 10 cases seen by him in Wilmington. Bellamy and J. B. Wright, of Lincolnton, were the first to recognize the disease in North Carolina, and did so before the appearance of Searcy's paper.

Moore has recently reported a case from Augusta, Georgia. Babcock's report to the South Carolina Board of Health aroused much interest in the matter. He described 12 typical cases.

At the 1907 meeting of the American Medical Association I reported a series of cases under the title of "Symmetrical Gangrene of the Skin," not recognizing their identity as pellagra. I suspected that malaria played a part in the causation of the skin lesion.

In Europe at the present time the chief seats of the disease are Northern and Central Italy and Roumania. The disease is endemic between 42° and 46° north latitude, 11° west and 26° east longitude of Paris. Several small foci are not included in this area.

In 1879 the number of cases in Italy was 97,855. About this time in Venetia, among 10,000 deaths, 500 were due to pellagra. Since 1881 the number has declined materially, due to the strenuous efforts on the part of the government in the more careful inspection of Indian corn and in the improvement of the general

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hygienic conditions. Next to Venetia stands Lombardy, with 300 deaths, then Emilia, with 200 deaths. Piedmont, Umbria, and Tuscany attribute over 5 per cent. of all deaths to pellagra. Neusser claimed that in Venice, in 1886, with a population of 36,588 natives, there were 1086 cases of pellagra.

In Roumania, in 1885, there were 10,626 cases; in 1886, 19,797 cases out of a population of 5,300,000. In 1892 there were 4500 cases; in 1898, 21,272 cases. Triller says that in 1906, in Roumania, there were 30,000 pellagrins. From 1893 to 1898 Sandwith reported 500 cases at the Kasrel Ainy Hospital in Cairo. In Algiers the disease is well distributed. Triller's idea of the extent of the disease is amazing. He claims that in certain parts of Italy, in 1906, from 30 to 50 per cent. of the population were affected; that in 1899 the number of cases in all Italy reached 73,000, which was more than 10 per 1000 of the rural population. Tuczek claimed that in 1884 there were 10,000 pellagrins in Italian institutions.

Pellagra has extended with great rapidity throughout the South during the past few years. It has been reported from North Carolina, South Carolina, Georgia, Alabama, and Texas. I have been able to find reports of 200 cases. Of this number, 88 are from the above-mentioned epidemic reported by Searcy, in Alabama, 25 are at present under the care of J. W. Babcock, at the State Hospital for the Insane in Columbia, South Carolina, 12 were reported by McCampbell from the Western North Carolina Hospital for the Insane, and 3 from the North Carolina State Hospital for the Insane under the care of J. H. McKee. In all, 65 cases have been recognized in North Carolina; of this number, 25 have occurred in New Hanover County, with an approximate population of 40,000.

The disease appears essentially in two forms. In one, the chronic, we have the typical picture described by the Italians: Symmetrical erythema, especially of the exposed portions of the body, which appears usually in the spring associated with stomatitis, diarrhea, often some gastric disturbances, and followed by cord symptoms of various kinds, and finally by mental disturbances of

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varying degree, and cachexia. As summer advances the symptoms usually disappear, only to reappear with the following spring. With each recurrence the impression on the nervous system becomes more indelible, the cachexia more marked, and the skin more atrophic and discolored. The average duration of these cases in Italy is five years, though not infrequently the duration is twenty years. One of Tuczek's cases was diagnosticated pellagra in 1851, and died of bronchopneumonia in 1887.

Mrs. W. (Dr. Thomas M. Green's case), aged thirty-four years. Nothing of note in her family or personal histories except malaria. In the spring of 1906 she suffered with indigestion and diarrhœa. One month after the occurrence of these disturbances an erythema appeared on the backs of her hands, around both eyes, and on the back of the neck, much which resembled sunburn. During the summer her condition became improved. In the spring of 1907 she suffered a recurrence similar to what she had had in 1906. In May, 1908, her condition was as follows: A poorly nourished, cachectic woman, appearing much older than her real age. Situated on her forehead symmetrically were two areas of desquamating erythema. These areas were symmetrical in position, shape, and size. Between the two areas was a vertical strip of normal skin. Lesions of like character surrounded both eyes. On the upper lids the lesions were more recent, and were covered with crusts with a weeping surface beneath. The lesions extended into the anterior nares. The lesions were present below the eyes, and there was present brownish pigmentation, indicating their greater age. On the backs of both hands, extending from the finger nails to the middle of the forearm, there existed the moist lesions, which were covered with crusts. Over the knuckles and the tip of the styloid the condition was exaggerated, the surface being entirely raw. The lesion was present on the anterior surface and the palms, and like the posterior, except that the skin of the palms was simply erythematous with exfoliation. Near the margins of the arm lesions there was considerable brownish pigmentation. On the back of the neck was a much older lesion, which had caused a tawny discoloration. Posteriorly, this lesion was about two inches

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in width, but became narrowed as it extended anteriorly. From the two sides the lesion lacked two inches of meeting in front, forming a sort of collar. The labia were affected with a moist lesion. She had had an obstinate diarrhea and stomatitis. Physical examination was negative. The urine contained a small amount of albumin and granular casts. The blood showed a simple anemia of moderate degree and no leukocytosis. The differential leukocyte count showed no abnormal variations.

When first seen this patient presented the picture of profound melancholia. As her condition became worse her mental state changed to one of wildest mania, making it necessary to forcibly restrain her to prevent her leaving the bed. She never spoke, but made incoherent cries, and presented the spectacle of great fear. She refused nourishment when it was offered, but would take it if it were left at her side. Her whole body was in a condition of tonic spasm. In her mania the crusts had been rubbed off of the diseased areas, and the bleeding surfaces made a loathsome patient. She died about three weeks after her admission to the hospital. The autopsy showed general fatty changes in the liver, with slight increase in connective tissue around the bloodvessels. There was also a slight round-cell infiltration. The capsule was thickened. The kidneys showed moderately advanced interstitial changes. The spleen and adrenals were negative. The changes in the cord were inconsiderable and indefinite save for obliteration of the central canal. The case was not sufficiently chronic to have produced very characteristic nervous changes.

X. (Dr. J. B. Wright, Lincolnton, N. C.), female, white, aged eleven years. This patient belonged to the better class. The course of the disease was typical, except that she had four attacks in six years, dying in the fourth, when seventeen years of age. The four attacks began each time in March.

The erythematous recurrences of pellagra usually occur yearly, and almost invariably in the spring. One of my earlier cases presented an interesting deviation from this rule.

A. R., white, married, aged forty-six years, a grocer. His family and previous history were both negative. He was a mag-

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nificent specimen of a vigorous, robust man. The condition was ushered in with high fever without prodrome. This fever was probably an attack of malaria. It was followed by a profuse sweat and apparent recovery. One week later his strange actions attracted his wife's attention. He would ring his own door bell and appeared dazed, making many absurd mistakes in his business. This was followed by a general apathy. He suffered from no delusions or hallucinations, and there were no sudden outbursts. After six months there appeared in June, 1905, two spots of erythema on the backs of his hands. He suffered at this time from diarrhea and had no control over his bowel or bladder. He would lie quietly in bed. His speech was low and hesitating and his gait unsteady. His habits were most untidy. The erythematous areas, which were of the dry variety, cleared up, and after desquamation the skin was soft and smooth. His mental condition, however, did not improve. In October, 1905, he was admitted to the James Walker Memorial Hospital, in Wilmington, N. C. At this time the erythema had reappeared and extended from the ends of his fingers posteriorly to two inches above the wrist joint. The lines of demarcation on the two hands were symmetrical in the most minute detail. There was also a lesion on the external surface of the right leg. This lesion involved the lower third of the lower leg and the external surface of the foot. It had caused considerable destruction of the skin. There was a corresponding lesion of smaller size and of the dry variety on the left leg. This patient's blood presented a remarkable condition. For three weeks the blood was loaded with tertian malarial parasites, and his chart, which was carefully kept, showed no rise of temperature. At the end of this time the temperature suddenly rose to 105° and the blood showed the preponderance of quartan parasites. From that time his temperature chart showed remarkable variations, and the blood showed the preponderance of first one variety of parasites and then another. In the spring of 1906 this patient had his third attack, and died in an epileptiform seizure.

From the beginning a case may be classified as acute or chronic without difficulty. The following is a typical case:

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D. J., female, colored, aged twenty-one years, a field hand. She was troubled in February, 1908, with a bloody diarrhea, indigestion, globus hysterica, and leucorrhea. After three months the erythema appeared in the "snuff box" of the right hand. This spot desquamated before the corresponding lesion of the left hand appeared. In June, 1908, she was admitted to the James Walker Memorial Hospital. On the backs of both hands, from the finger tips upward, was found a dry exfoliating lesion of the skin, which was darker than the normal. The upper margins of the two lesions made oblique lines extending from a point at the middle of the forearm internally downward and outward to within one inch of the wrist joint. The same line was carried out anteriorly, and the lesion in this respect was unusual. There were small, round, symmetrical areas on the upper lids near the inner canthi, and at the outer canthi smaller areas. Small areas were found at the angles of the mouth. On the tops of the feet over the ankle joint and also on the tops of the toes were present similar lesions. The patient had never gone without shoes. There was no history of stomatitis. In July the lesions subsided and the patient was discharged. This was her first attack.

It remains to be seen what will be the course of some of our chronic cases which have survived the first attack, or even the second, and are entitled to be counted chronic cases.

The acute or fulminating variety of pellagra, called by Lombroso "typhoid," differs widely from the Italian form. It runs a course usually of from three weeks to three months, and invariably ends fatally. The patients usually die before any marked nervous changes appear other than those usually incident to a profound acute toxemia. Over 50 per cent. of the cases in the Southern States are of this variety. Belmondo and a number of other Italian observers claim that this form is never primary; that it is always an exaggeration of a regular recurrence of the chronic form of the disease.

B. B. (Dr. R. H. Bellamy, Wilmington, N. C.), aged thirty years, white, married. When first seen the disease had existed for several weeks. The patient was much emaciated, and was suffering from

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an obstinate diarrhea and stomatitis, which suggested ptyalism. On the backs of the hands and the posterior surface of the forearms there were found moist exfoliative skin lesions. The same lesion was present on the back of the neck. In the beginning the patient thought he had simple sunburn. In all situations the chief characteristic is absolute symmetry. After a few weeks this patient died of exhaustion.

V. S. (Dr. W. J. H. Bellamy, Wilmington, N. C.), negro, female, aged twelve years. In February there appeared at the outer canthi of the eyes two small red symmetrical areas. These areas extended over the forehead. It next appeared beneath the angles of the jaws, and from there around the neck, meeting behind. Soon after the appearance of the skin lesions there developed a severe stomatitis. The next step in the disease was the appearance of the erythema on the backs of the hands, including the fingers. From the beginning there was always an obstinate diarrhea. In May she came under my care. At this time the whole face was covered with the lesion, which appeared like an old burn and had caused much contraction, producing thereby an ectropion of both lower lids and distorted the mouth. She was unable to open her mouth wide enough to permit any examination, and she also was unable to close her eyes completely. Both hands and both lower thirds of the forearms were covered anteriorly and posteriorly with the moist lesion. Conjunctivitis soon developed, and there was marked subconjunctival edema. The feet were edematous, and later (several weeks) the lesion appeared on the dorsal surfaces. The knee jerks were absent. Skin sensation normal. Pupils reacted normally to light and accommodation. The patient complained constantly of cold. Her appetite at first was good, but later she refused all nourishment. The affected skin was exfoliated, leaving perfectly raw surfaces. The patient passed 80 round worms during the three weeks she was under observation. She died of exhaustion in June.

In Italy the disease is confined to the peasantry almost exclusively. There poverty and pellagra are interchangeable terms. With us the disease is affecting all classes alike. Of the 25 cases

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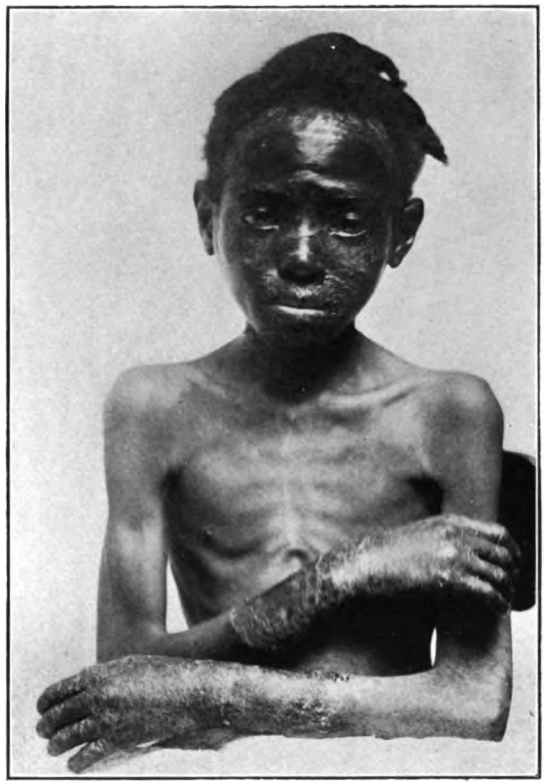

Fig. 1.—V. S., aged twelve years; acute pellagra; death in first attack.

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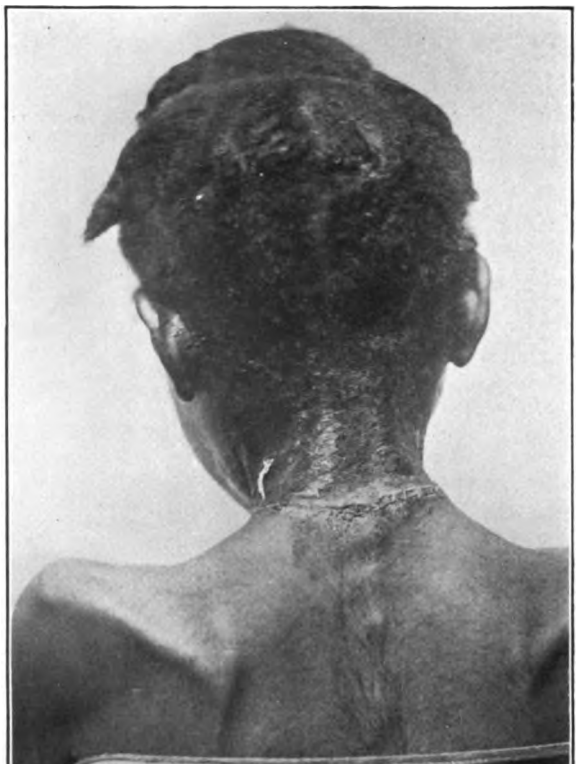

FIG. 2.—Same case, showing lesion of neck and ears.

FIG. 3.—Same case, showing the lesions of the hands.

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which I have studied, in none was there a history of a preëxisting chronic affection as tuberculosis. As a rule, the victims were previously well nourished and robust until affected with pellagra.

We have had occasion to consider whether the disease is infectious or contagious and also whether it is a family disease. The case just referred to was one of two members of the same family simultaneously affected. The other member was a younger sister, who has the chronic form and recovered from the first attack. I have seen four children of the same parents affected simultaneously. Two of these patients died suddenly in the first attack. The other two have passed from our observation.

A prodrome is acknowledged by all pellagralogers. This prodrome usually begins about Christmas time, and is characterized by indefinite symptoms, as anorexia, or voracious appetite, pain and sensation of distention in the epigastric region, frequently diarrhea, though oftentimes constipation, insatiable thirst, or the reverse. Later there appeared headache, which is chiefly occipital, pain in the back and the neck, hyperesthesias, dizziness, muscular weakness, and uncertainty of motion. Roussel considered sensations of dryness and burning in the mouth and heat in the stomach as the first symptom of the disease. In most respects these symptoms present nothing peculiar, but we have learned that when our patient complains of headache, vertigo, sensations of weakness especially in the lower extremities, together with diarrhea, to become suspicious of pellagra, especially if the season be late winter or spring. Theodori claimed that these pre-pellagrous symptoms existed four weeks before the appearance of the erythema, but in many cases the period is much prolonged.

The erythema almost invariably makes its appearance in the spring. In one series it appeared as early as February and as late as July. The greatest number developed in April and May. The uncovered portions of the body, as the hands, face, and neck, are more apt to be affected, and in all cases are first affected. Authorities have always differed in regard to the role played by insolation in the causation of the disease. From our experience we are disposed to reject the idea. The disease usually appears so early

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in the spring that the sun's rays are of little consequence. Those of our cases with the lesions of the skin of the feet were in most cases adults who were not in the habit of going without shoes. Further, the skin over the sternum is often affected. Some writers think that the actinic rays are responsible for these lesions of the exposed parts of the body.

The backs of the hands are invariably the first situation of the lesions. This occurred in all of our cases. The lesion in this position is very distinctive, and usually furnishes sufficient evidence for a positive diagnosis. The first appearance is often mistaken for sunburn; no two things could be more alike. The lines of demarcation between the normal and diseased skin are absolutely symmetrical in position and direction. The points of greatest intensity are usually the skin over the knuckles and tip of the styloid processes. The lesions usually begin at the finger nails and extend upward to the junction of the middle and lower thirds of the forearm. The general appearance is usually that of diffuse redness without swelling, but in some cases the swelling is added and makes its appearance before the erythema. The condition terminates in dry exfoliation, or there may appear blebs which rupture, leaving raw surfaces which soon become covered by crusts. The appearance of the skin after healing is variable. It may be soft and velvety like an infant's skin, but there is always a tendency to the deposition of pigment, which increases with each recurrence. In three of our cases the lesion extended to the palmar surfaces, and in this position we always noted the soft, smooth condition of the skin after the lesions disappeared.

After the hands the face is most apt to be affected, and we found this much oftener in women and children than in men. Perfect symmetry is maintained here just as in the hand lesions. Often the first appearance on the face are two small spots at the outer canthi of the eyes or the angles of the mouth. In several cases there were two spots of erythema on the forehead, with a narrow strip of normal skin exactly in the midline separating them. Sometimes the lesions assume the butterfly shape of lupus. Small areas below the lower lids are frequent. We have seen the lesions on

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the back of the neck with a maintenance of the same symmetry, and oftentimes there is lateral extension of these lesions which may form a collar meeting in front. The next point of selection is the tops of the feet. It occurred in one-third of our cases. In an acute case the erythema had much the appearance of an acute lymphangitis, beginning over the crest of the tibia and extending downward over the tops of the feet to the toes. As before mentioned, the erythema may appear over the sternum. The labia pudendi is a frequent seat of the lesion, even in children. With healing there often is associated contraction of the skin, as in superficial burns. In photograph No. 1 marked contraction of the skin of the face is shown with resulting ectropion and contractions of the skin of the upper and lower lid, which made complete closure of the lips impossible. In the dry lesions exfoliation usually occurs after three or four weeks. The resulting pigmentation varies from a slight yellowish tinge to a brown or chocolate coloration, depending on the number of the attacks.

The stomatitis appears usually after the appearance of the skin lesion, sometimes long before, and again may be entirely absent. It is a very characteristic symptom, and seldom fails to appear at some stage of the disease. It often suggests ptyalism. The gums are usually swollen and intensely red. The tongue is red and often denuded of epithelium, giving the characteristic "bald tongue." The papillæ are enlarged, with furrows between. The patient complains of the sensation of a salt taste and burning in the mouth. Salivation is very frequent. In all of our cases the mouth was affected in some degree.

The stomach is variously affected. In some cases the hydrochloric acid is normal in amount, while in others it is absent. Pyrrhosis, eructations, vomiting, anorexia, or bulimia, together with great thirst, have been emphasized by various writers.

One of the most unfailing symptoms is an obstinate diarrhea which is most intractable. Sometimes it assumes a dysenteric character with violent colic. This intestinal condition is considered by many as a neurosis.

In the acute form of the disease the nervous disturbances are no

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more than ordinarily occur in any profound toxemia. It is difficult to say at what stage the nervous disturbances appear in the chronic variety. In one of the cases previously referred to a robust man developed mental disturbances of a peculiar sort, and had been in an institution for the insane a number of months before the first appearance of the pellagrous erythema. I recently discovered the first attack of pellagrous erythema in a woman who for several years had been in a hospital for the insane with a diagnosis of melancholia. Can it be that apart from pellagrous insanity the insane are predisposed to the disease?

The first nervous symptoms manifested are usually paresthesias. Chief among those are usually mentioned itching of the backs of the hands and the back, occasionally also of the lower extremities. The patients under my care often complain of pain of a burning character in the right or left scapular region. This latter is a very common symptom often referred to. This burning sensation often occurs in the epigastrium, the hands, and the arms, and is supposed to be the cause of the tendency among pellagrins to throw themselves into the water whenever occasion presents itself. Numbness and formication are often complained of. One of the most constant complaints is cold extremities, and it is no unusual sight to find our patients in midsummer huddled before a fire trying to keep warm.

The vertigo of pellagra is a symptom much emphasized by the Italian observers, and was seldom wanting in our cases. It is responsible for many falls. By some it is considered merely a simple lapse of consciousness. According to the careful observations of McCampbell, of the Western North Carolina Hospital for the Insane, the psychic manifestations were preceded by a period of anxiety and worry, with the attachment of undue importance to trivial things. There is often a simple slowness of ideas. A question is replied to long after the patient seems to have forgotten it. There often occurs an irritable depression, with aversion to any activity. Later, there develops disturbances of perception with development of hallucinations. Judgment may be disturbed, giving rise to delusions which usually are painful and

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depressive. These delusions are usually more of fear and suspicion. Our hospital cases were a great care because of their persistence in disarranging the bedclothes, attempting to get out of bed, and especially in their total disregard of all cleanliness. We were disposed to consider at first this latter as viciousness, but soon learned that it was mental hebetude. Refusal of food and suicidal tendencies were frequent in our series. As the disease approaches its close the patients become wildly delirious, and it is usually necessary to resort to the restraining sheet. They frequently cry out in a wild incoherent, fashion, and in their ravings it is impossible to prevent damage to the skin lesions, with the result that large areas are left raw and bleeding. The patient in such cases presents a horrid sight.

A condition is often observed somewhat resembling tetany, with paroxysmal tonic contractions and contractures of the upper and lower extremities in the semiflexed position. The gait is simple paralytic, occasionally paralytic spastic, but never ataxic. There is something characteristic, however, in the unsteadiness and seeming lack of confidence with which these people shuffle along. Static ataxia is occasionally mentioned. Incoördination is seldom observed, and then only in the upper extremities. Tremor of the arms, head, and tongue sometimes occurs.

Definite epileptiform attacks with loss of consciousness are occasionally observed, having the character of a cortical epilepsy. One of our patients died in such an attack.

The skin sensibility is very irregular. The sense of taste is usually normal. The faradocutaneous sensibility is often increased. Sometimes there is decrease of tactile and thermal sense. Owing to the altered condition of the patient's intellect and the resulting unreliable character of the replies to questions, any investigation of sensation is unsatisfactory.

The skin reflex is usually normal. The pharyngeal reflex is not infrequently decreased. The tendon reflexes are quite variable, oftentimes normal, often increased to the point of an intense clonic contraction. Much less frequently they are decreased or absent. It sometimes occurs that the reflex is increased in the

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lower extremity and normal in the upper extremity. In Tuczek's 300 cases one-half had increase in the knee-jerk up to the rapid patellar clonus, even with the slightest percussion of the tendon. In 35 cases there was dorsal clonus and increase of the tendon reflex of the upper extremity. In 8 cases the knee-jerks were absent, and in the remaining it was partly lessened and partly normal. Differences in the two sides were often noted.

Among the vasomotor disturbances is described a vasoparalytic dilatation of the veins and capillaries and not infrequently edema. The skin lesions are usually counted as trophic disturbances. Pellagra is usually described as a feverless disease. The temperature chart showed that during the active stage of the disease there is invariably a rise of temperature ranging from 99.5° to 102° F.

The urine is alkaline and deficient in phosphates. When the toxemia is intense there is the usual albuminuria.

What is called by most writers the third stage is characterized by a general atrophy of the whole body: the subcutaneous fat disappears and the patients present the appearance of advanced cachexia. Profound weakness necessitates rest in bed. The bladder becomes paralyzed and the diarrhea is uncontrollable. Death is usually directly attributable to myocardial degeneration and its consequences.

In the very chronic cases there is frequently an acute terminal exacerbation, and it is this condition that the Italians have in mind when they speak of the "typhoid" pellagra. Evidently they are not familiar with the acute pellagra so common with us. This explains Belmondo's statement that "typhoid" pellagra is never primary, but merely an exacerbation of a very chronic case. It is in this terminal condition that the delirium is so wild. Tuczek claims that at autopsy fever could be found to have been due to a bronchopneumonia. In our experience there is just as much cause for fever in pellagra as there is for fever in a large superficial burn which has become infected.

"Pellagra sine pellagra," or pellagra without skin manifestations, is a point of contention. Some observers consider erythema or atrophy and pigmentation of the skin essential for the diagnosis.

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Babcock, in South Carolina, at the present time has a number of such cases under his care. He says these patients have stomatitis, diarrhea, and disturbances of the nervous system. My own experience is too limited to make the diagnosis without reserve. However, I have under my care several cases which I have diagnosticated "pellagra sine pellagra." One of these cases gives the following history:

Mrs. O., aged sixty-three years. Five years ago she was seized with a peculiar attack which was thought to be apoplectiform. She recovered from the attack, but has never been quite well since then. She has had repeated obstinate attacks of stomatitis and diarrhea. At present she is bedridden on account of profound weakness. She has an obstinate diarrhea and is quite emaciated. There is no erythema. She replies to questions with marked tardiness. The patellar reflex is absent. The pupils react to light and accommodation in rather an exaggerated degree. I am convinced that in many of the so-called cases of "pellagra sine pellagra" there is or has been a very mild erythema which is not visible at the time when the patient comes under observation. All pellagralogers state that the extent of the erythema is no index to the severity of the disease.

Recently there came under my care a white woman, aged forty-six years, mother of eight children, with a negative family history and a negative previous medical history. In July of this year she had an acute gastritis which was quite severe in character. Soon after this she developed diarrhea, and later a severe stomatitis.

She made a slow recovery. During convalescence the palms of her hands and the soles of her feet desquamated. On close inquiry I elicited the fact that early in her sickness her hands had a purplish cast which attracted attention at the time. Careful examination showed the last remnant of the upper border of what must have been the pellagrous erythema. Certainly, the lesion was very inconspicuous. At present this patient is a great sufferer. The pain in her hands and feet is constant, and her hands show beginning contractures. She is unable to comb her hair or button her clothes. She walks with great unsteadiness, and says she has the sensation

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of walking on roller skates. Her station is poor. The pupils react sluggishly to light and accommodation. The knee jerks are greatly exaggerated. Except for increased emotion, the mental condition is little changed. She is much emaciated and is losing ground steadily. Mistakes in the diagnosis are often made, owing to the fact that the order of appearance of the symptoms may be entirely reversed. I have seen stomatitis, diarrhea, and all the mental changes appear a year before the first indication of the skin lesion. It is very likely that in "pellagra sine pellagra" sooner or later there will be skin manifestations.

The pathological changes that occur in pellagra depend largely on the chronicity of the affection. As in ergotism, the most definite changes are in the spinal cord. These changes are just what the symptoms would indicate. In ergotism the posterior root zones are affected, but this does not occur in pellagra. The latter disease shows affections of the lateral rather than the posterior columns. Allbutt says the weight of the disease falls on the crossed pyramidal tracts; the direct cerebellar tracts are not affected; the cells in the anterior horn are deeply pigmented. The lesions of the posterior columns occur chiefly in the cervical and upper dorsal regions, while those of the lateral columns affect the middle and lower third of the dorsal region.

In the eight autopsies which Tuczek reports the posterior columns were degenerated in seven. Of this number, in one the degeneration was limited to Gall's column and a medial strip in Burdach's column. In five the degeneration extended throughout the whole length of the cord. In one the lesions were found in the cervical portion alone. In four the dorsal region was the chief seat of the disease. In five the crossed pyramidal tracts were affected. In the first the affected areas extended from the upper cervical to the upper dorsal, with degeneration of the cells in the anterior and posterior horns in the middle and lower cervical. There was atrophy of cells in the anterior horns and Clarke's column of the upper dorsal. There was atrophy also of the anterior root fibers. In the second the degeneration of the crossed pyramidal tracts was more intense in the dorsal regions, but extended as high upward

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as the middle cervical and as low as the upper lumbar. In the third case the lesion extended throughout, but was especially marked in the dorsal portion. In the fourth case the lesion was confined to the dorsal portion, and in the fifth degeneration was massive and limited to the middle cervical portion. Tuczek further found abnormal pigmentation similar to senile changes, especially of the ganglion cells. He considered the presence of hyperemia, anemia, and edema together with the occasional inflammatory affections of the central nervous system coverings, as well as obliteration of the central canal of the cord—all a part of many chronic affections of the nervous system, and while present in pellagra, they should not be considered characteristic.

The changes in the brain are inconspicuous except for occasional fatty degeneration or calcification of the intima of the smaller vessels and pigmentation in the adventitia.

In one of my earlier cases, in which, as was above mentioned, an unusual malarial infection was thought to play a part, the tissues were carefully studied by Dr. H. A. Cotton, Superintendent of the New Jersey State Hospital for the Insane. Dr Cotton reported in part: "The cortex showed no reaction that could be called pathological. The topography was unaltered and the elements of the cortex were not affected by the malarial infection. There was probably an excess of pigment in the nerve cells, which could be accounted for by the patient's age. The bloodvessels appeared a trifle thickened, but the fixed cells did not seem altered or to have proliferated at all, and the vessels were not increased in number. There was no evidence of general paralysis or other organic affections, and no evidence of cerebral syphilis. The plasmodia were very easily seen in the blood stream and in large numbers. The Weigert preparations for medullated fibers did not show any degeneration, but in the Marchi preparations there was a scattered degeneration in both cortex and cord. These scattered degenerations probably only meant that there was some toxic effect of the malarial infection. It was only a very moderate degeneration and of little significance."

The stamp on the nervous system can only be discovered after

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the disease has existed for years. As all of our cases had run only very short courses, the pathological changes were not marked.

Pigmentation and fatty degeneration of the thoracic and abdominal organs occur. To this is added atrophy chiefly of the organs innervated by the vagus—the heart, kidneys, spleen, intestines, liver, and lungs. There is atrophy also of the coats of the intestine, with occasional hyperemia and ulceration of the lower portion.

The pathological changes seem to be slow degenerations rather than inflammation, and is probably not essentially progressive in character.

In 1776 the Socita patriotica di Milano offered a prize for a solution of the question of the etiology of pellagra and a means to combat it. Marzari, in 1810, suggested the relationship of corn in the etiology. Strombia, the elder, and Marzari claimed that the corn was deficient in nitrogen because it was sown late and gathered green. Forcher and Roussel claimed that it was a disease of the corn which produced the toxin. Lombroso emphasized the fact that healed pellagrins when given good corn food became affected again. Pellagrins seem to show a susceptibility at certain seasons of the year for even the smallest quantity of pellagrous material. Corn ordinarily contains many microörganisms, and certain parts of the kernel make an excellent nidus. Lombroso concedes that the best corn is often most diseased, and that the trouble is rather in some noxious agent which uses the corn as a host. The *acarus farinæ* was supposed to play an important role by making a point of entry into the kernel through which the noxious agent entered. Moisture is one of the greatest sources of trouble. The corn, besides being gathered green, is not always carefully handled, and is in consequence poorly protected from moisture. It is said that the custom among the Italian peasantry of preserving pollenta for days and even weeks, allowing fermentation to occur, is the chief source of danger.

In studying the etiology of the series of cases occurring in my section, I was impressed with the fact that practically all of the corn used by the people came from Ohio and Virginia, and in neither of these States had pellagra ever been reported. While by no means

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ready to accept the corn as the source of the disease, I am of the opinion that if there is any connection the noxious agent must appear after the shipping of the corn. Many Southern observers attach importance to the heating of the corn which so often occurs in the cars before they are unloaded. Atmospheric conditions probably play an important part.

Last spring I collected three samples of corn which I had reason to suspect of containing pellagrous material. These samples were placed in my laboratory, which during the summer months is a damp place. After an absence of several months I returned to find everything covered with mould. It occurred to me that the conditions were favorable for the development of any fungus, and I sent the three samples to the North Carolina Experiment Station. Dr. F. L. Stevens furnished me with the following report:

No. 1.

	Per cent.
Penicillium	30
Aspergillus	60
Syncephalstrum	10

No. 2.

	Per cent.
Aspergillus	70
Penicillium	20
Chetomium	5
Syncephalstrum	5

No. 3.

	Per cent.
Mucor	5
Penicillium	45
Aspergillus	50

Dr. Stevens considered each sample a good average grade of corn and suitable for food.

Various moulds have been brought forward as the etiological factor. Most common among these is the penicillium crustaceum, which was not found in any of the corn consumed by my patients.

Lombroso's pellagrozein is claimed to produce pellagra in the lower animals. While there are toxic symptoms produced, they are not sufficiently typical to be counted as pellagra. Two organisms have been isolated from corn. The first is a short bacillus producing an

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orange-yellow growth on agar-agar, staining by Gram's method and forming no spores. The cultures are abundant and gelatinous. It does not produce gas. This organism is non-pathogenic. The second bacillus produces spores. On agar-agar it produces faint round colonies, which also grow on glycerin agar. It liquefies gelatin. This bacillus, too, is non-pathogenic to rabbits. It corresponds with the Bacillus mesentericus or with the Bacillus maidis of Peltauf and Heider. This organism was isolated from corn meal and corn bread as well as from the feces of pellagrins. It is claimed that this organism is also found in the feces of the normal individual.

Majocchi, in 1881, described the Bacillus maidis as a very motile organism which he found in the bodies of pellagrins in the beginning of the disease. Cuboni found the organism in diseased corn, and claimed that it resisted the boiling temperature. Peltauf and Heider claimed that the Bacillus maidis developed like the Bacillus mesentericus or the potato bacillus, and that it was not always present in the feces of pellagrins. They considered the organism non-pathogenic, but an alcoholic extract of the corn meal containing them caused narcosis, paralysis, and death in mice. Babes and Sion produced symptoms resembling pellagra in various animals by the injection of this bacillus. Ballardine, working with the sparisorium maidis, was able to produce in man gastro-intestinal symptoms, but his results were not conclusive.

Tizzoni isolated an organism from the blood, cerebrospinal fluid, organs, and feces of pellagrins. With this organism he produced, without question, the disease pellagra in rabbits and guinea-pigs. Tizzoni also found this same organism in two out of nine samples of suspected corn. In these experiments Tizzoni infected twenty-eight animals. Of this twenty-eight, four were infected by mouth; three with pellagrous material, and one with a culture from the blood of a pellagrin. The remaining twenty-four were infected subcutaneously. Of this latter number, eight with the blood of a pellagrin, twelve with an organism isolated from the feces of a pellagrin, and four from corn. The remaining twelve were infected with pellagrous material. The disease so

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produced ran an acute course of from sixteen to fifty days and a chronic course from eighty-seven to one hundred and five days. Autopsy showed lesions of the skin and also of the abdominal organs of a hemorrhagic type. The changes in the nervous system were varied and indefinite.

Tizzoni and Panichi, in a previous communication, claimed that they could only produce the disease experimentally when, in addition to their inoculations, the animals were fed on corn food. Lombroso was the chief supporter of the chemical theory of the decomposition of corn as the cause of pellagra. He experimented with great quantities of diseased corn containing aspergillus, rhizopus, Bacillus maidis, and acari. The dogs on which he experimented were fed milk, bread, and flesh, and on the administration of his preparation from the corn the temperature rose and the weight decreased. There usually appeared psychic and muscular torpidity, and in some cases anesthesia and tremor. Out of ten cases, seven had diarrhea, and in one there was an erythema. In all, there were appearances in the dog considered the counterpart of pellagra in man. The feeding of bad corn meal caused erythema, stupor, and dysphagia. He prepared from the corn an oil, a fluidextract, and a tincture. He gave to twelve individuals daily the corn tincture, and in eight cases there was itching of the back and the face and loss of weight, in five cases diarrhea, and in five cases desquamation. The appearances were not exclusively those of pellagra, and the result is therefore not conclusive.

Neusser believed that corn contained a non-toxic glucoside produced by the Bacillus maidis, and that in the intestine this body is decomposed, producing a toxic substance, but that this only occurs when the bowel is already in a diseased condition. There is not sufficient ground for this view.

Antoniu reported seven cases fed on diseased corn in addition to meat and cheese. Of this number several became pellagrous, but even this observation is not counted as conclusive because of the possibility of the victims having been previously pellagrous.

Erba's pellagrozein, prepared from bad corn, caused convulsions, increased reflexes, narcosis, and anesthesia. He noted that in summer his product was more toxic.

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Babcock, quoting Sambon, says that the association of pellagra with the eating of maize is too universal to be ignored, and that, based on pathology and symptomatology, there is good ground for the hypothesis that pellagra is a protozoan disease allied with trypanosomiasis and syphilis.

I have isolated from the blood of a pellagrin in the active stage of the disease a bacillus which produced spores, is decolorized by Gram's method, and liquefies gelatin. Like the bacillus of Peltauf and Heider, it belongs to the potato bacillus group. It is probable that it is also the same as the very actively motile organism described by Majocchi. My observations on this organism are not sufficient for a detailed report at this time; however, it is altogether probable that Peltauf and Heider, Cuboni, Majocchi, Tizzoni, and myself have found at different times the same organism.

The prognosis of pellagra in this section is very grave. We have many evidences of the fact that when an infectious disease first appears in a country it is much more malignant than later on. This is well illustrated in the lower mortality among the Japanese troops in their recent war with Russia from beriberi (about 3 per cent.), while in a new country this mortality often reaches 60 per cent. The same condition obtains in measles and yellow fever outbreaks. Fulminating pellagra is an utterly hopeless disease. The death rate of the chronic pellagra is very variable, and it is too soon for us to report definitely on this subject. So far we have not observed a case older than three years.

The treatment of acute pellagra is of no avail. We have reason to suspect that the epizoötic meningitis among horses is due to the same cause as pellagra in man. If the above-mentioned organism is the cause of pellagra and the horse is susceptible, we have reason to hope that the serumtherapy will soon come to our assistance. It has been shown that the blood of a healed pellagrin is curative in a guinea-pig experimentally inoculated with pellagrous material.

In the chronic cases it is possible that a cure may result from the removal of all corn food and general tonic treatment. Atoxyl is much vaunted just now, but in our hands the results have not been so brilliant as reported by the European pellagralogers.

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In closing, I wish to acknowledge my gratitude to my fellow practitioners of North Carolina for their generous assistance in collecting data for this paper.

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DISCUSSION.

DR. HENRY A. COTTON: I was much interested in these cases of pellagra, and through the courtesy of Dr. Wood I was able to examine the brain of one of his cases. Unfortunately, the findings were negative except for a scattered degeneration of fibers in the cortex and cord by the Marchi method. There was apparently no definite reaction in the cortex, as the nerve cells and neuroglia were normal.

In looking up the literature of this subject, one is struck with the meagre description of the mental phenomena, also of the lack of any definite histopathology of the nervous system. Dr. Wood has given a very good description of the mental symptoms as observed in his cases.

Recently a report upon pellagra was made by the Staff of the State Insane Hospital in South Carolina (in the *American Journal of Insanity* for April, 1908), but the report is incomplete, and loses much of its value by a lack of accurate clinical observations of the mental symptoms, as well as the absence of any histopathological data. These gentlemen were inclined to consider their cases more nearly related to Egyptian pellagra than to the Italian form, and they spoke of the association of "hook-worm" disease with pellagra as being significant.

Adriana, in 1885, published a very important article regarding this disease. At first it was confounded with a form of malaria. He claims that 5.5 per cent. of the cases become insane. There were present muscular spasms, anxiety, apathy, and stupor. He mentions the researches of Lombroso and others, who found an oily resin, and an azotized alkaloid, called pellagrozenia, which caused the toxic symptoms. The only changes reported by this author, were softening of the white and gray matter of the spinal cord, and granular myelitis. He claims that two and eight-tenths to one thousand population were pellagrous.

Clerici described pellagrous insanity as accompanied by stupor and loss of memory, and Procopiu describes it as acute or chronic delirium, not unlike delirium tremens; the usual termination is dementia. The most prominent symptoms are dermatitis, diarrhea, and depression.

According to Bianchi, the later researches of Ceni and others have demonstrated that fungi on maize were penicillium glaucum and aspergillus fumigatis. The chronic subacute forms of mental trouble apparently were due to the former, and the "pellagra typhus" form was due to the latter. The disease is caused by means of toxins set free in the gastro-intestinal canal, although Bianchi is inclined to think the latter form, pellagra typhus, is caused by infection rather than intoxication.

Déjèrine found parenchymatous neuritis, and in the brain were found thickening, turbidity, and adhesions of the meninges, atrophy, and induration of

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the cerebral substance, increase in the subarachnoid fluid, profound alteration in the cerebral cells, and increase of neuroglia in the chronic forms. The anatomical pathological alterations were thus very similar to progressive paralysis or tabes, and often degeneration of the posterior columns of the cord in the cervical region.

Babes and Sion found lesions resembling tabes, and these lesions most frequent in the cervical segment.

According to Bianchi, the nervous and mental phenomena dominate the picture, and he speaks of the chronic and acute forms. The chronic forms show general depression, progressive wasting, melancholia, confusion, slow dementia, parasthesia, alteration in motility, ataxic gait, and gastro-intestinal disturbance, obstinate diarrhea; contractures and subsulti were absent. Reflexes were exaggerated.

In the acute varieties, rapid elevation of the temperature, neuromuscular excitement, subsulti, contractures, muscular rigidity, more marked exaggeration of the reflexes, and more pronounced mental confusion, with phases of exaltation, more like pellagra typhus.

It would seem, from these accounts, that we are dealing with a severe intoxication and accompanied by delirium of various degrees of severity. The symptoms noted by the various observers are somewhat vague. The pathological anatomical findings were also indefinitely reported, so that it leaves one in doubt as to the true nature of the disease, that is, as regards the brain lesions.

As Dr. Wood speaks of "pellagra sine pellagra," I would like to mention a class of cases recently described by Adolph Meyer, under the term "central neuritis," in which the mental symptoms are not unlike those shown in pellagra. Associated with this stupor or anxious delirium, we have rigidity, peculiar muscular spasms or jactitations, and obstinate diarrhea. They all terminate fatally. The etiology of central neuritis is obscure, but the process is probably due to an intoxication, and the histopathology has been definitely described, and can easily be diagnosticated by a competent neuropathologist. I merely mention this as having some bearing upon the etiology and pathology of pellagra, as well as showing a similarity in process and termination.

I do not mean to say that the two diseases are similar, but that they both speak for a similar pathological process. I do not agree with the authors mentioned above, that the disease processes resemble paralytic dementia or tabes, or is in any way a similar process. It is a common error to class an obscure and unexplained lesion of the nervous system with general paralysis. But now we have a definite idea of the histopathology of general paralysis, and certainly, such a disease as pellagra should not be confused with it. I hope to examine more material from these cases, and look for definite lesions to be found.

Dr. Louis A. Duhring: I have listened to the paper of Dr. Wood with great interest for several reasons; in the first place, the disease pellagra has

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been presented to us by the reader of the paper in a new light. He has portrayed the symptomatology of this disease in quite a different aspect from well-known writers in Europe. So far as our knowledge extends, the disease has been practically almost unknown in the United States. Everyone knows that there are two distinct sets of symptoms with this diesase. The one which attracted observers one hundred or more years ago had as the most prominent symptom lesions of the skin. Having had the opportunity of studying in the wards of the City Hospital of Milan, Italy, many years ago, I found that the pellagra patients were congregated in the wards for cutaneous diseases. The disease was regarded in those days chiefly as a cutaneous affection. True, it was said other cases which we also regard as pellagra are to be found in quite different kinds of institutions, some in insane asylums, so that even in those days, it may be noted, the nervous and mental conditions were also recognized. There did not seem to be at that time a great deal of information to be derived from observing the cases in Milan. They were regarded much as cases of lepra might have been, that is, to some extent neglected as concerned investigation. Since that time much work has been done in Italy and in other countries. The disease has been regarded by most European writers in quite a different light from that of the reader of this paper, especially as a chronic disease, having the train of symptoms much as described by Dr. Wood. The reader of the paper has spoken of the disease running an acute course, and he tells us that in the United States we have a form of the disease which has markedly acute symptoms, has a rapid course, and that a large number of the cases have been fatal. This is in strong contrast to the cases in Southern Europe and in Egypt, the majority of which pursue a chronic course. I am greatly impressed with the statement that many of the cases described by Dr. Wood occurring in South Carolina and Alabama have been rapidly fatal. This is a new and important aspect of the disease. That is not at all the usual course. Of 400 tabulated cases occurring in Cairo, some years ago, there was, if I remember aright, a mortality of only 5 per cent. The point of most interest stated by Dr. Wood is that he has presented a new type of pellagra, running an acute course, frequently fatal, and with somewhat different symptoms from those observed by most of the earlier foreign writers. He has presented an important matter for the consideration of the physicians of the United States, especially those of the South.

In this zone of climate, I think it may be stated, pellagra does not occur endemically. I do not think we have had any case in Philadelphia, and only a few have been encountered in New York. I saw one case about twelve years ago in New York, through the courtesy of Dr. Sherwood, of Brooklyn. The patient was an Italian immigrant, who had contracted the disease in his own country. His food had consisted largely for a long time previously of maize. The opinion has long prevailed throughout Europe that the cause of the disease is defective or spoiled maize.

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It is now well known that the cutaneous lesions are often, if not usually, really a minor symptom of the disease, and I can well understand the statement of the reader of the paper that he believes he has seen cases without the cutaneous symptoms. Others have made the same observation. Ten or fifteen years ago, in writing an article on pellagra for my work on *Cutaneous Medicine*, I came to the same general conclusion. The most important symptoms are those of the cerebrospinal system.

The etiology of the disease was not this evening touched upon to any extent, but it would be most interesting to know the opinion of the author on this point. It may be here remarked that another case occurred in New York about twenty years ago, and in this instance peculiar cutaneous lesions were very marked. The man died three months later from the effects of the disease. There are many other points about pellagra of extreme interest, and I believe that this paper will be the means of bringing the whole subject before the profession of the United States, especially in the South, in some parts of which, at least at the present time, the disease exists to an alarming extent. It is well known that in Europe it does not go north of a certain latitude. It is almost unknown in Germany, in Norway, and in Sweden. I believe from what I, myself, have seen that the disease is capable of having almost as many different manifestations throughout the body as is either tuberculosis, leprosy, or syphilis. These manifestations have, as yet, only been touched upon by most writers in the literature of the subject, and not to the extent they deserve to be described.

DR. M. B. HARTZELL: The disease is so well marked and the symptoms so characteristic that it does not seem likely that it has been formerly overlooked; we must, therefore, regard it as a new disease in this country. If maize is the chief cause, it is remarkable that it should have appeared only within the last ten or fifteen years, because maize has been the food of the Southern people for a very long time. It would seem as if there were some other element in its etiology than the use of spoiled maize.

DR. HOBART A. HARE: I should like to ask Dr. Wood whether there are cases on record in which the persons had not been fed on Indian corn. I think the general opinion is that the maize which produces pellagra is not necessarily primarily diseased, but that it has been gathered while very green and has sweated, very much as hay "sweats," and in that way there is produced a factor productive of the symptoms.

DR. WOOD, closing: Manson thinks that the diagnosis of pellagra is only justified when the patient has been eating Indian corn, and that symptoms of pellagra without the history of corn consumption would justify the diagnosis of pseudopellagra. It is probable that corn is the host of the noxious agent, and also that Tizzoni's organism, which has been isolated from the blood, feces, cerebrospinal fluid, and organs of pellagrins as well as from corn, is the etiological factor. I have found this organism in the blood.

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Lombroso thinks that the corn which is responsible for the trouble has become deteriorated, but again he says that the best corn is often the most diseased.

In my own section, where pellagra is abundant, the corn comes from Ohio and Virginia, and in neither of these States has there been any reports of outbreaks. I am inclined to think that whatever deterioration may occur in the corn takes place after its shipment. Too many cases have been reported in individuals who do not eat corn extensively for me to be willing at this time to accept the corn theory without some reservation.

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APPENDIX.

REPORT OF THE COMMITTEE ON ENTERTAINMENTS OF THE LAYING OF THE CORNER STONE AT THE NEW HALL OF THE COLLEGE OF PHYSICIANS, TWENTY-SECOND AND LUDLOW STREETS, PHILADELPHIA, APRIL 29, 1908.

This invitation

THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

REQUESTS THE HONOR OF YOUR PRESENCE

AT THE CEREMONIES OF THE

LAYING OF THE CORNER STONE

OF THE NEW BUILDING

TWENTY-SECOND AND LUDLOW STREETS

ON WEDNESDAY THE TWENTY-NINTH OF APRIL

AT FOUR O'CLOCK

R. S. V. P.

DR. CHARLES H. FRAZIER,
1724 Spruce Street.

was sent to all the Active and Associate Fellows of the College, to all who had contributed to the present building fund, and to many who had been contributors to various funds in the past. Invitations were also issued to the presidents of the Philosophical Society, Historical Society, Academy of Natural Sciences, the Wistar Institute, and to the representatives of the daily papers.

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Acceptances were received from about one hundred and sixty of the Active Fellows; of the Honorary Fellows, Dr. A. Jacobi, of New York, was the only representative in attendance.

The following is a copy of the order of ceremonies:

CEREMONIES AT LAYING OF THE CORNER STONE

OF THE NEW BUILDING

OF THE

COLLEGE OF PHYSICIANS, PHILADELPHIA

APRIL 29, 1908

PRAYER BY RIGHT REV. OZI W. WHITAKER, D.D.

SHORT ADDRESS BY THE PRESIDENT, DR. JAMES TYSON

LAYING OF THE CORNER STONE BY DR. S. WEIR MITCHELL

After a prayer by Right Rev. Ozi W. Whitaker, D.D., the following address was made by Dr. James Tyson:

The Homes of the College of Physicians.

Organized on January 2, 1787, and chartered in 1789, the College of Physicians of Philadelphia doubtless took its name from its ancient and honorable prototype, the Royal College of Physicians of London. Its first meetings were held, in the parlance of its minutes, "at the University," meaning the then new University of the State of Pennsylvania, which was incorporated by the Legislature in November, 1779, and was endowed with the sequestered properties and rights of its predecessor, the College of Philadelphia, and with other confiscated estates. The College of Philadelphia was the lineal descendant of the "Academy and Charitable School in the Province of Pennsylvania," incorporated July 13, 1753, by the proprietors, Thomas and Richard Penn. The school was originally suggested by Benjamin Franklin, in 1743, and was actually started in 1750, during which year the pupils were taught in private houses. But as early as December, 1749, the Trustees had procured a lot near the southwest corner of Fourth and Arch Streets, on which was a large building, which they altered to suit their purposes. It was completed in 1751, and henceforth known as "The Academy." By 1755 it had acquired such renown and its curriculum was so advanced that the proprietors granted an additional charter May 14, in which the title was changed to "The College, Academy, and Charitable School of Philadelphia," reciting and

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confirming all the franchises of the first, with the further power of conferring degrees.

Here it was, in the old Academy Building, on Fourth Street, that the first meetings of the College were held, and in this building was also born the Medical School of the University, in 1765. In the meantime, however, after ten years, the rights and properties of the College of Philadelphia were restored to it and the University of the State of Pennsylvania ejected from the premises, although the latter was permitted to retain some of the confiscated properties. Two years later, in 1791, the two were united under the title of the University of Pennsylvania. The College held its meetings in the Academy Building for nearly five years.

The next home of the College was the Hall of the Philosophical Society, 204 South Fifth Street, where it met for the first time on December 10, 1791, and continued to meet there until July 1, 1845, or more than fifty-three years. Toward the latter part of this time the Fellows began to be restless as to the place of meeting, and various steps were taken to secure another hall. At one time, in 1832, it was proposed to erect a building for the joint accommodation of several societies, and a proposition arose to form a Medical Hall Association of Philadelphia, to procure "an edifice suitable to accommodate the meetings of various associations, and be a convenient and safe deposit for their libraries and museums," but was rejected, it being deemed inexpedient for medical societies in their corporate capacity, either singly or jointly, to undertake the work. A room offered by the University of Pennsylvania was declined in September, 1840.

A committee of the College, appointed for the purpose November 3, 1840, reported that the cost of a proper building for the College would exceed $15,000, and although a plan was approved and many liberal subscriptions promised, it did not seem possible to raise a sufficient sum, and the committee asked to be discharged. On October 5, 1841, a committee, directed to ascertain whether a suitable room could be obtained in a hall recently purchased by the Philosophical Society (Chinese Museum, Ninth Street south of Chestnut), reported that no definite information could be obtained. On April 4, 1843, the Philosophical Society increased the rent of the room occupied by the College to $50 a year. On March 4, 1845, a committee reported that an airy, well-lighted room on the third floor, separate and distinct from the rest of the building of the Mercantile Library, could be secured at an annual rent of $185. With commendable thrift the College directed its committee, June 3, to engage the room at $175 a year, from July 1, 1844, and have it suitably fitted to accommodate the library and the sessions of the College. This move met with hearty approval and stimulated greatly the interest of the Fellows. The last meeting in the Philosophical Society room was held July 1, 1845.

After the balance of rent due was paid there remained only $16.43 in the

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treasury, while the cost of fitting up the "Hall" of the College, as it was now called for the first time, was $280.42½. But the Fellows contributed liberally, raising by subscription $213 and by sale of old bookcases $9.45, leaving a deficit of $57.97, which the Treasurer was ordered to pay. On July 1, also, the entrance fee was increased to $15 and the annual contribution made $5.

As already stated, the new movement stimulated the interest of the Fellows, and the meetings were better attended and other activities were aroused. Seven years was, however, the life of the College in the room rented of the Mercantile Library Co., and in 1852 the "Picture House" of the Pennsylvania Hospital, 820 Spruce Street, built for the accommodation of Benjamin West's picture of "Christ Healing the Sick," was rented for $250 a year. Here the College continued to meet until its removal to its own building, at Thirteenth and Locust Streets, in March, 1863, eleven years later.

Thus the College has had five abodes since its organization in 1787: first, the Academy Building on Fourth Street, for nearly five years; second, the Hall of the Philosophical Society, for fifty-three years; third, the Mercantile Library, for seven years; fourth, the "Picture House" in the Pennsylvania Hospital Grounds, for eleven years; and fifth, its own hall, at Thirteenth and Locust, from March, 1863, to the present time. So that the new building which we are met to formally initiate this afternoon will be the sixth home it has occupied in a life of one hundred and twenty-one years. Justly proud of our existing Hall at Thirteenth and Locust Streets, the first of its kind in this country erected solely for the advancement of the healing art and representing a great achievement for its day, it is too contracted to serve the purposes of the College at the present day, to say nothing of the future, and the new building will be a larger, more complete, and more imposing but still simple edifice, thoroughly in accord with the history and traditions of the College.

The foundations being well under way, the stage has been reached at which we are ready for the laying of the corner stone, which will now be done by our distinguished and dearly beloved Fellow and ex-President, Dr. S. Weir Mitchell.

At the conclusion of Dr. Tyson's address, Dr. S. Weir Mitchell placed in the corner stone the copper box, already sealed, which contained

A copy of the last report of the Library Committee.

A copy of the last TRANSACTIONS of the College of Physicians.

A copy of the list of the members of the Building Committee.

A copy of the Philadelphia *Press*, dated April 29, 1908.

Photograph of the present Hall of the College of Physicians, taken by Wm. H. Rau.

As the corner stone was being lowered, Dr. Mitchell spoke as follows:

I am honored by the wish of the President and the Fellows with the privilege of laying the corner stone of the College of Physicians of Philadelphia.

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This ancient institution has a distinguished and fertile history of one hundred and twenty-one years, and thus is as old as the Constitution of the United States. Therefore, with pride in the memory of our illustrious dead, and with glad confidence in the promise of a future no less productive of good to mankind, I now fulfil my grateful task and declare laid the corner stone of this new home of the greatest and most progressive of the professions.

Respectfully submitted,

CHARLES H. FRAZIER, M.D.,
Chairman.

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FIFTH TRIENNIAL REPORT OF THE REPRESENTATIVE OF THE COLLEGE ON THE STATE QUARANTINE BOARD.

At the close of my three years' term (the fifth) as representative of the College on the State Quarantine Board, I present, as usual, my report. I have, however, very little of interest to communicate beyond the routine business of the service.

There have been several cases of beriberi treated at the Station at different times, all occurring among Orientals, and without a death. Eight of these cases were taken from a steamship from Sourabaya, Java, the *Knight St. George*, on October 31, 1905, nine days after she had reached her dock, the disease not being recognized before that time; these patients did not return to the city, but were taken on board the *Knight St. George* as the vessel passed the station outward bound, November 15. Another case of beriberi was removed from the steamer *Andova*, from Iquique, Peru, January 10, 1906; and still another from the steamer *Indra*, from Madras, India, reaching Philadelphia June 30 of the same year. Finally, seventeen cases of beriberi were received at the State Quarantine from the ship *Spithead*, which arrived at this port from Sourabaya, November 9, 1906.

In an "Address on Sanitation in the Japanese Army and Navy," delivered at the University of Pennsylvania in 1906,[1] by Baron K. Takaki, Surgeon-General of the Imperial Japanese Navy, the speaker claimed that beriberi, or kakke, as it is called in Japan, had been banished from the Japanese Navy, to which it had been a terrible scourge, entirely incapacitating in its ravages among the men vessels from service, by a change in the food ration. He attributed the disease to a deficiency of proteid and an excess of carbohydrate in the food. This condition was met in the new ration by the substitution of barley for a part of the rice in the old, barley containing more proteid (nitrogen) than rice; later, bread and biscuit took the place of rice and barley. This view does not necessarily negative the prevailing theory in regard to beriberi that it is a germ disease; if true, it shows merely that the germ may be successfully resisted with a suitable diet, just as it is claimed the bacillus of tuberculosis may be successfully fought by a *regime* in which nitrogenous diet figures largely. It would seem worth while that a change of diet in the merchant

[1] University of Pennsylvania Medical Bulletin, 1906–1907, xix, 246.

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marine of the East were tried; an obstacle is the greater expense of barley, bread, and biscuit over the rice, so abundantly produced in the Orient.

Two cases of smallpox were treated successfully at the Station last fall, the victims reaching there after a curious sequence of events. On September 28, 1907, the steamer *Vienna*, from Sourabaya, arrived off the State Quarantine Station. It was reported that the vessel had reached the Capes of the Delaware five days before with a man suffering from smallpox on board, the man dying immediately after the Breakwater was reached; that the body of the man had been cremated at the United States Quarantine Station at that place; that the vessel had been disinfected, being detained three days; and that the five men who had been closely associated with the sick man, and had been employed in the cremation of his body, had been left behind at the United States Quarantine Station. With this report the State officials, discovering no sick on board and finding that every person on the vessel bore marks of recent vaccination, or presented evidence of successful vaccination, permitted the vessel to proceed to her wharf at Philadelphia, where she began unloading her cargo. On October 1 the men who had been taken off at the Breakwater returned to their ship, which was still unloading in Philadelphia, coming to the city by rail, without, therefore, passing through the hands of the State Quarantine officers. Two cases of smallpox developed among these men, upon which the ship was ordered to the State Quarantine Station; here the ship was again disinfected and sent back to Philadelphia; the crew, however, was detained until October 23, when, no other cases of disease occurring, they were released; the sick men meanwhile being placed in the hospital, from where they were discharged on their complete recovery. These incidents are related to emphasize the danger of suspects leaving quarantine before a sufficient number of days have elapsed to make it certain that none of them had acquired the disease. Only eight days had passed from the date of the death of their companion when the sailors who had been detained at the United States Quarantine Station reached Philadelphia. The rule of the State Quarantine requires that suspects in cases of smallpox should be detained not less than fourteen days, unless evidence is present of successful vaccination.

In July, 1905, the Board experienced a serious loss in its personnel in the retirement of Dr. Benjamin Lee, due to the abolition by the Legislature of the office of Secretary of the State Board of Health, in virtue of which office Dr. Lee was *ex-officio* a member of the State Quarantine Board. Dr. Lee had acted as Secretary of the latter Board twelve years—since its organization in 1893—filling an office for which he was well qualified by his tireless energy, wide knowledge of hygiene, and literary accomplishments. Under the new law passed by the last Legislature creating a State Commissioner of Health, Dr Lee's place as a member of the Quarantine Board is held by Dr. Samuel G. Dixon, the incumbent of that new office, a well-known Fellow of the

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College and President of the Academy of Natural Sciences. Another gap in the membership of the Board occurred recently in the death of Chas. F. Giller, Esq., whose appointment to the Board as representative of the Maritime Exchange I noted in my last report. He was an active and very popular member of the Board. He has been succeeded by Richard J. Watson, Esq., a gentleman very prominent in the foreign fruit trade of the port of Philadelphia.

Governor Stuart, the new Chief Magistrate of Pennsylvania, in arranging his administration, retained Dr. H. D. Heller in the office of Quarantine Physician, who is now serving his third term.

The excellent physical condition of the buildings and grounds has been maintained and improvements made in the laying of asphalt walks and the introduction of gas from a gas plant in the city of Chester. The water supply of the Station is at present obtained from a well on the grounds, but arrangements have been made by which water as well as gas will be procured from Chester. When this is accomplished, the Station will be more secure from the danger of fire.

The State Legislature, at its last session, appropriated to the Board, to cover expenses of the two years 1907 to 1909, $42,500; this sum is $500 in excess of that appropriated for the preceding two years.

Appended are tables relating to the work done at the State Quarantine Station during each of the three years covered by this report. The following is a summary for the whole period:

Number of vessels inspected and passed	4,965
Number of passengers inspected and passed	87,383
Number of officers and men inspected and passed	148,639
Number of vessels detained for disinfection	50
Number of officers and men detained for disinfection	182
Number of medical and surgical cases examined and treated on ship-board	621

RICHARD A. CLEEMANN.

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APPENDIX.

June 1, 1905, *to May* 31, 1906.

Number of vessels inspected and passed	1,635
Number of passengers inspected and passed	27,923
Number of officers and men inspected and passed	47,350
Number of vessels detained for disinfection	16
Number of officers and men detained for disinfection	115
Number of medical cases examined and treated	76
Number of surgical cases examined and treated	96

June 1, 1906, *to May* 31, 1907.

Number of vessels inspected and passed	1,748
Number of passengers inspected and passed	34,852
Number of officers and men inspected and passed	50,703
Number of vessels detained for disinfection	14
Number of medical cases examined and treated	112
Number of surgical cases examined and treated	94

June 1, 1907, *to May* 31, 1908.

Number of vessels inspected and passed	1,582
Number of passengers inspected and passed	24,608
Number of officers and men inspected and passed	50,586
Number of vessels detained for disinfection	20
Number of men detained for disinfection	67
Number of medical cases examined and treated	136
Number of surgical cases examined and treated	107

JUNE 3, 1908.

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ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1908.

The number of volumes in the Library is	77,603
General Library	60,562
Lewis Library	13,515
S. D. Gross Library	3,309
Obstetrical Society Library	217
There are also:	
Unbound "Reports" and "Transactions"	9,115
Unbound "Theses" and "Dissertations"	22,681
Unbound Pamphlets	67,122

No duplicates are included in the above number of volumes, excepting 2212 retained as "Reserves," consisting chiefly of second copies of important periodical publications.

In addition to these there are now on hand, but to be disposed of by sale or exchange, 2854 duplicates.

There have been received during the year 3015 volumes, 8366 pamphlets, and 22,218 numbers of various medical periodicals; 40 of the new books received were written or edited by Fellows of the College.

The increase in the number of volumes is 1987.

The Library receives by purchase, in exchange, or as gifts, 630 periodical publications—183 American and 447 foreign. Twenty-nine new subscriptions were added during the year.

The following list shows how these are distributed and credited:

	American.	Foreign.
Henrietta Rush Fales Baker Fund	0	30
William T. Carter Fund	1	23
John D. Griscom Fund	4	37
S. D. Gross Library Account	0	3
William F. Jenks Fund	2	30
Journal Association	1	12
	8	135

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Carried forward	8	135
Oliver A. Judson Fund	0	6
William V. and John M. Keating Fund	0	1
William W. Keen Fund	3	19
Library Endowment Fund	4	123
J. Ewing Mears Account	0	1
Weir Mitchell Fund	1	34
Philadelphia Medical Society Fund	0	4
Lewis Rodman Fund	0	12
John F. Weightman Fund	0	4
Caspar Wistar Fund	4	14
By Purchase	2	17
In Exchange	82	63
Editors	67	14
Publishers	12	0
	183	447

In addition we receive current numbers of 154 American, and 82 foreign medical, pharmaceutical, and dental periodicals, sent to the Library chiefly by the Editors of:

American Journal of the Medical Sciences.
Medical Council.
Monthly Cyclopedia and Medical Bulletin.
Therapeutic Gazette.
University of Pennsylvania Medical Bulletin.

We exchange publications with the following schools of medicine:

University of Basel.	University of Kiel.
" Berlin.	" Königsberg.
" Bern.	" Lausanne.
" Bonn.	" Leipzig.
" Breslau.	" Marburg.
" Erlangen.	" Rostock.
" Giessen.	" Strasburg.
" Göttingen.	" Upsala.
" Griefswald.	" Würzburg.
" Halle.	" Zurich.
" Heidelberg.	
Faculty of Medicine of Bruxelles.	
" " Nancy.	
" " Paris.	
" " Rio de Janeiro.	
" " Toulouse.	

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Nine hundred and forty-eight inaugural dissertations have been received during the year.

Forty-four books were presented by the following authors or editors:

Dr. A. P. C. Ashhurst.
Dr. J. M. Batten.
Mr. W. K. Bixby.
Dr. J. W. Bovée (editor).
Dr. J. H. Claiborne (editor).
Dr. W. A. Newman Dorland.
Dr. A. R. Edwards.
Dr. Seneca Egbert.
Dr. William Ewart.
Mr. Horace Fletcher.
Dr. Alfred Gordon.
Dr. G. M. Gould.
Dr. H. A. Hare.
Dr. Lucien Howe.
Dr. A. A. Hubbell.
M. Jeannel.
Dr. H. A. Kelly.
Mr. Charles R. Lanman (editor).
Mrs. Millicent W. Miller.
Dr. C. S. Minot.
Dr. William Osler.
Dr. G. A. Piersol (editor).
Dr. L. S. Pilcher.
Dr. Pires de Lima.
Dr. John B. Roberts.
Dr. C. E. de M. Sajous.
Dr. T. M. Satterthwaite.
Dr. Henry Lee Smith.
Dr. E. A. Spitzka.
Dr. Frederico Torralbas.
Mr. Edward Trust (editor).
Mr. David T. Watson.
Dr. Joseph Walsh (editor).

Twenty volumes were sent by the publishers, at the request of the following authors and editors:

Dr. A. L. Benedict.
Dr. E. P. Davis.
Mr. Horace Fletcher.
Dr. R. M. Goepp.
Dr. G. M. Gould.
Dr. H. A. Hare (editor).
Dr. T. B. Holloway.
Dr. Edward Jackson.
Dr. M. K. Kassabian.
Dr. H. A. Kelly.
Dr. W. W. Keen (editor).
Dr. H. R. M. Landis (editor).
Dr. W. T. Longcope (editor).
Dr. Charles P. Noble.
Dr. W. L. Rodman.
Dr. Jay F. Schamberg.
Dr. G. E. de Schweinitz.

There were 407 donors to the Library during the year ending November 1, of whom the following presented 25 volumes or more:

Mrs. Eliza Kirkbride	195 volumes.
Dr. J. J. Taylor	156 "
Dr. William G. Spiller	146 "
Mrs. M. M. Dunmire	122 "
Mrs. D. F. Greenwald	78 "

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Dr. Hobart A. Hare	76 volumes.
Dr. George W. Norris	76 "
Dr. Frederick P. Henry	42 "
Dr. John B. Roberts	39 "
Dr. Horatio C. Wood	38 "
Dr. Arthur V. Meigs	36 "
Dr. A. O. J. Kelly	32 "
Dr. John B. Shober	30 "
Dr. John K. Mitchell	29 "
Dr. Joseph Leidy	28 "

Gifts of books were also made by the publishing houses of:

P. Blakiston's Son & Company	16 volumes.
Broadbent Press, New York	1 "
F. A. Davis Company	11 "
Lea & Febiger	52 "
(42 of these were bound volumes of the *Medical News*)	
J. B. Lippincott Company	18 "
C. V. Mosby, Chicago	1 "
W. B. Saunders Company	32 "
Frederick A. Stokes Company, New York	5 "
William Wood & Company, New York	3 "

The Library has received large gifts of pamphlets and numbers of periodicals from the following gentlemen:

Dr. Lewis H. Adler, Jr.	Dr. E. B. Hodge, Jr.	Dr. J. B. Roberts.
Dr. O. H. Allis.	Dr. W. W. Keen.	Dr. G. G. Ross.
Dr. John Aulde.	Dr. A. O. J. Kelly.	Dr. C. E. de M. Sajous.
Dr. W. H. Bennett.	Dr. E. Lindauer.	Dr. G. E. de Schweinitz.
Dr. S. Solis Cohen.	Dr. M. J. Lewis.	Dr. E. A. Shumway.
Dr. E. P. Davis.	Dr. Morris Longstreth.	Dr. E. R. Siter.
Dr. W. A. N. Dorland.	Dr. O. J. McCarthy.	Dr. W. G. Spiller.
Dr. C. W. Dulles.	Dr. Edward Martin.	Dr. Louis Starr.
Dr. A. A. Eshner.	Dr. A. V. Meigs.	Dr. J. M. Swan.
Dr. C. H. Frazier.	Dr. C. K. Mills.	Dr. J. J. Taylor.
Dr. T. H. Fenton.	Dr. J. K. Mitchell.	Dr. C. F. Taylor.
Dr. L. F. Flick.	Dr. S. Weir Mitchell.	Dr. H. R. Wharton.
Dr. J. P. C. Griffith.	Dr. T. R. Neilson.	Dr. J. William White.
Dr. S. McC. Hamill.	Dr. C. P. Noble.	Dr. A. C. Wood.
Dr. H. A. Hare.	Dr. G. W. Norris.	Dr. H. C. Wood.
Dr. G. H. Harlan.	Dr. C. A. Oliver.	Dr. William Zentmayer.
Dr. F. P. Henry.	Dr. F. D. Patterson.	Dr. M. W. Zimmerman.

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And from:

P. Blakiston's Son & Company. W. B. Saunders Company.
J. B. Lippincott Company. John Wyeth & Brother.

The account of the books purchased by the various "Funds" is as follows:

	Volumes purchased.	Cost.	Volumes bound.	Cost.
Henrietta Rush Fales Baker Fund	81	$184.93	31	$23.35
Luther S. Bent Fund	27	43.14	1	.60
William T. Carter Fund	72	197.39	21	19.70
John D. Griscom Fund	84	235.74	27	19.40
William F. Jenks Fund	80	252.88	21	24.55
Oliver A. Judson Fund	14	41.67	5	2.20
William V. & John M. Keating Fd.	12	16.28	7	5.50
William W. Keen Fund	62	159.80	15	10.65
Library Endowment Fund	145	600.09	..	..
Weir Mitchell Fund	50	168.92	..	..
Elizabeth K. Newcomet Fund	3	8.06	2	1.75
Philadelphia Medical Society Fund	5	8.81	..	..
Lewis Rodman Fund	33	164.48	14	16.20
John F. Weightman Fund	24	69.81	5	4.50
Caspar Wistar Fund	46	143.75	17	12.95
Total	738	$2295.75	166	$141.35
Special Accounts:				
Fd. for completing Files of Journals	201	$421.45	..	..
Journal Association	24	130.14	..	..
New Book Fund	46	72.20	..	..
J. Ewing Mears Account	21	64.83	9	$10.20
S. D. Gross Library Account	56	173.12	11	16.70
Total	348	$861.74	20	$26.90

George B. Wood Fund:
For Library supplies, stationery, etc. $412.39

All accessions of books and the more important pamphlets have been catalogued, and each volume has been accessioned and shelf-listed; 4640 pamphlets have been examined, 1196 retained and classified; 11,988 volumes have been re-accessioned.

Revision of Catalogue:
Number of cards revised, typewritten, examined, and alphabeted. . . 8213

1025 books have been bound.

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We have received $139.32 by the sale of duplicates, and there is a credit to our exchange account with the New York Medical Book Company of $274.49. $30.85 has been collected from fines.

There have been 12,166 visitors to the Library—5110 more than during the preceding year.

22,409 books have been consulted, besides those taken from the shelves by Fellows of the College, of which no record can be kept—2363 more than during 1907.

5064 books have been taken out—502 more than reported last year.

Through the liberality and generous efforts of Dr. W. W. Keen, there have been added to the Library during the past year thirty-three rare and valuable medical works, of which twenty-one were printed in the fifteenth century, and known as incunabula; the titles of these are as follows:

Arnoldus de Villa Nova. De arte cognoscendi venena. n. p. ca. 1473. 4to.

Arnoldus de Villa Nova. Tractat von Bereitung von Bruchung der Wein. ca. 1473. n. p. 4to.

de Manfredi, Hieronimus. Liber de homine. (Bononiæ, 1474.) 4to.

Guy de Chauliac. Nel nome de dio comeza. Venensia, 1480. 4to.

Ægidius (Carbolensis), Petrus. Incipit liber magistri Ægidii de pulsibus metrice compositus. Paduæ, 1484. sm. 4to.

Matheolus (Perusinus). Tractatus clarissimi philosophi et medici Matheoli Perusini de memoria. (Rome, ca. 1484.) sm. 4to.

de Tornamira, Joannes. Incipit clarificatorium Joannis de Tornamira super nono Almansoris cum textu ipsius Rasis. (Lugduni.) 1490. sm. 4to.

Mesue, Joannes. Mesue cum additionibus Francisci de Pedmontium. Venetiis, 1491. F°.

Ardoynis, Santes de. Liber de venenis. Venetiis, 1492. F°.

Celsus, Arelius. Medicinæ liber primus incipit. Venetiis, 1493. F°.

de Manliis de Bosco, Joannes. Luminare majus. Romæ, 1494. F°.

de Ketam, Joannes. Fasciculus medicine in quo continentur. Venetiis, 1495. F°.

Tractatus de vino et eius proprietate. Roma, ca. 1495. 4to.

(Rare; not mentioned by Hain or other bibliographers.)

Burlæus, Gualterus. (Walter Burley). Preclarissimi viri Gualteri Burlei anglici sacre pagine professoris excellentissimi super artem veterem Porphyrii et Aristotolis expositio. Venetiis, 1497. F°.

Coradinus. De morbo quem gallicum. (n.p., 1497.) sm. 4to.

Leonicenus, Nicolaus. De epidemia quam Itali morbum gallicum. Mediolani, 1497. sm. 4to.

Avenzohar (Abumeron). Incipit liber theicrisi dahalmodana vahaltadabir. Venice, 1497. F°.

Sermoneta, Joannes. Questiones super libros affrorismoris Ypocratis. Venetiis, 1498. F°.

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del Garbo, Dinus. Expositio Dini Florentini super canonis Avicene. Venetiis, 1499. 4to.

Brunschwig, Hieronymus. Liber de arte distillandi. Strasburg, 1500. 4to.

Tartaretus, Petrus. Totius philosophiæ necnon metaphysicæ Aristotelis expositio. Volume II. Lyons, 1500. 4to.

There are two other works, which, while not incunabula, are worthy of special mention:

Avicenna. (Libri V canonis medicinæ.) Arabic Text. Romæ, 1593.

(An extremely handsome copy printed in Arabic on tinted paper, with the original parchment binding, stamped with the seal and crest of the Duke of Altemps.)

Dioscurides. Codex Aniciæ Julianæ.

(A magnificent edition of the photograph facsimile of the Dioscurides codex, in two large folio volumes, published at Leipzig in 1906.)

Dr. Keen, to assist him in the purchase of these books, by personal solicitation obtained over $1100 from the following gentlemen, whose names have already appeared in this report under the "List of Donors:"

Mr. George A. Armour.	Mr. Harold Pierce.
Mr. Alex. Blair Thaw.	Mr. Joseph G. Rosengarten.
Mr. W. W. Frazier.	Mr. Carroll Smyth.
Mr. Henry C. Lea.	Dr. De Forest Willard.
Mr. J. N. Pew.	Dr. W. W. Keen.

There is a small unexpended balance, which will be used for the purchase of books of the same class.

Another service of great value to the Library of the College was the effecting of an exchange with the Faculté de Médecine de Paris, through the efforts of Dr. W. W. Keen, Dr. William Osler, Dr. A. J. Magnin, and Dr. Louis Landouzy, the Dean of the Medical Faculty, by which we receive the forty or fifty volumes of the *Paris Theses*, published each year, in exchange for our TRANSACTIONS.

The importance of making some effort toward completing our files of the *Thèses de Paris* was so great that an appeal was made to certain Fellows of the College for subscriptions; this was generously responded to, and $1415 were collected for the purpose. Of this amount Dr. Keen obtained $315, and the Librarian the remainder. This money has been most judiciously expended by Dr. Keen while in Paris, and his purchases will complete our files from 1882 to 1907—more than 850 bound volumes.

The binding of the *Theses* delayed their shipment until a date too late to be included in this report under "Accessions to the Library;" therefore, this

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valuable collection will be included in the report for 1909, and the subscribers' names added under the list of "Donors to the Library."

While President of the College, Dr. Keen obtained by personal solicitation, or by letter, a majority of the exchanges that we have with foreign Universities; and in consequence a large number of valuable inaugural dissertations have been received each year. While abroad this past summer, Dr. Keen has again taken up the matter of exchanges, and has communicated with twenty-five foreign Universities; the replies are now being received. Six, that have accepted, are added to this year's report, and it is expected that quite a number more will be added during the coming year.

There have been three changes during the year in the Library staff, which have considerably hampered the revision of the catalogue and the classification of the unbound pamphlets, but the work is now proceeding satisfactorily.

A certain appearance of confusion and disorder in the Library is rendered inevitable by its crowded condition and the want of a suitable workroom; but we hope for a striking contrast in this respect in the new quarters, preparations for moving into which during the coming summer are already being made.

The accompanying balance sheet of the Treasurer shows the receipts and expenditures.

The current expenses of the Library, aside from the purchase of books, for the coming year are estimated at $7047.59.

GEORGE C. HARLAN,
Chairman.

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REPORT OF THE COMMITTEE ON THE MÜTTER MUSEUM, 1908.

THERE have been fewer additions and contributions to the collections of the Mütter Museum than usual during the past year, owing chiefly to the approach of the moving of the College to its new building at Twenty-second and Ludlow Streets. The quarters allotted to the Museum are ample and more accessible, as they are on the ground floor, so that visitors will not have to climb the many stairs which have been a great drawback heretofore. The plans adopted by the committee for the arrangement of the display of the number of invaluable pathological specimens will be found to furnish opportunity and facility for all who may desire to make use of the Museum for practical study. During the past year the collections have been used by teachers of pathological osteology to many classes of students, and the Pathological Society held its annual meeting in the Museum Hall. The regular Mütter lecture was given on December 11 by Dr. George W. Crile, Professor of Clinical Surgery at the Western Reserve University. The subject of his address was "Surgical Anemia and Resuscitation." It was closely listened to by a large and appreciative audience.

The committee desire to have a complete set of copies of the Mütter lectures and photographs of the several lecturers from the time the Lectureship was established, and any information bearing upon this will be thankfully received.

Respectfully submitted,

GEORGE MCCLELLAN,
Chairman.

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LIST OF PAPERS: SECTION ON OPHTHALMOLOGY.

December, 1907.

Exhibition of a Case of Microphthalmos with Coloboma of the Optic Nerve, by Dr. G. Oram Ring.

Exhibition of a Case Showing a Growth at the Corneoscleral Margin, by Dr. G. Oram Ring.

Exhibition of a Case Showing a Foreign Body on the Iris, by Dr. G. Oram Ring.

Exhibition of a Case Showing a Corneoscleral Growth, by Dr. S. D. Risley.

Exhibition of a Case of Bilateral Exophthalmos, by Dr. S. D. Risley.

Exhibition of a Case of Bilateral Cicatricial Ectropion of the Upper Lids in a Patient with Extensive Lupus, corrected with Skin Flaps taken from the Arm, by Dr. G. E. de Schweinitz.

Exhibition of a Case of Epithelioma of the Inner Angle of the Eye Cured by *X*-rays, by Dr. G. E. de Schweinitz.

Exhibition of an Unusual Case of Keratoconus, by Dr. C. W. Le Fever. (by invitation).

Some Ocular Lesions Caused by Cerebrospinal Meningitis, with an Illustrative Case, and the Pathological Study of the Enucleated Eyeball, by Dr. G. E. de Schweinitz and Dr. C. M. Hosmer (by invitation).

Recurrent Vitreous Hemorrhages Occurring in Adolescence, with Report of a Case, by Dr. Frederick Krauss.

Exhibition of a Patient with Miliary Tuberculosis of the Palpebral Conjunctiva, by Dr. H. F. Hansell.

A Rare Form of Hereditary Cataract, by Dr. Wm. Zentmayer.

January, 1908.

Exhibition of a Patient Showing Extensive Bilateral Xanthelasma, by Dr. G. Oram Ring.

Exhibition of a Case of Successful Removal of a Foreign Body from the Iris, by Dr. G. Oram Ring.

The Subsequent History of a Case of Acquired Cataract in Childhood, by Dr. H. F. Hansell.

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Concerning the Obstetric Injuries of the Cornea, with Cases, by Dr. G. E. de Schweinitz.

Exhibition of a Patient with Microphthalmos, with extensive Colobomata and other Congenital Defects in Both Eyes, by Dr. Walter L. Pyle.

Exhibition of a Case of Bilateral Exophthalmos, by Dr. S. D. Risley.

Exhibition of a Case of Central Toxic Amblyopia presenting some unusual Features, by Dr. J. T. Carpenter.

February, 1908.

Exhibition of a Case of Chorioretinal Sclerosis, by Dr. Wm. T. Shoemaker.

A Case of Accessory Sinus Disease, with the Symptoms of an Osseous Tumor of the Orbit, by Dr. Burton Chance.

Attempts to Repair Extensive Destruction of the Orbital Tissues, following Disease of the Antrum, by Dr. Burton Chance.

A Case of Quinine Amaurosis, by Dr. Carl Williams.

A Case of Neuroma of the Orbit, by Dr. H. F. Hansell.

March, 1908.

Exhibition of a Case of Tubercle of the Iris, by Dr. Wm. Campbell Posey.

Exhibition of a Case of Electric Burn of Both Eyes, by Dr. Wm. Sweet, for Dr. H. F. Hansell.

Exhibition of a Case of Pseudoglioma of the Eye, by Dr. Edward A. Shumway.

Report of a Case of Paralysis of Extra-ocular Muscles in Exophthalmic Goitre, by Dr. W. C. Posey.

Report of a Case of Homonymous Hemianopsia in the Macular Regions, by Dr. W. C. Posey.

Two Cases of Interstitial Keratitis Associated with Cretinoid Conditions, by Dr. S. D. Risley.

Exhibition of a Case of Probable Congenital, Circumscribed Defect of the Choroid with Anomalous Pigment Arrangement, by Dr. Wendell Reber (by invitation).

Restoration of Contracted Socket, with Exhibition of a Patient, by Dr. Wm. Zentmayer.

April, 1908.

Spontaneous Rupture of a Reform Eye, by Dr. S. D. Risley.

The Ocular Symptoms of Ethmoidal Disease, with Exhibition of a Patient, by Dr. Frederick Krauss.

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Exhibition of a Case of Vascular Nevus of the Eyelids, Cured by Electrolysis, by Dr. S. D. Risley.

Gonorrheal Uveitis, with Exhibition of a Patient, by Dr. H. F. Hansell.

Exhibition of a Possible Case of Gonorrheal Uveitis, by Dr. W. C. Posey.

An Unusual Case of Congenital Squint, by Dr. W. C. Posey.

Exhibition of a Patient with Rupture of the Choroid, by Dr. Frederick Krauss.

October, 1908.

Exhibition of a Case Showing an Unusual Inflammation of the Cornea, by Dr. W. C. Posey.

Some Unusual Cases of Plastic Choroiditis, by Dr. G. E. de Schweinitz.

An Extensive Plastic Operation for Cicatricial Ectropion of Both Eyelids, with Exhibition of Photographs, by Dr. Wm. Zentmayer.

Exhibition of a Case of Amaurotic Family Idiocy, by Dr. Mary Buchanan (by invitation).

November, 1908.

Report of a Case Showing the Effects of Obstetric Injury of the Cornea in an Adult, by Dr. Burton Chance.

Again a Word as to the Weiss Reflex "Bogenstreife" in its Relation to Progressive Myopia, by Dr. B. Alex. Randall.

Exhibition of a Case of Growth in the Vitreous, Probably Arising in the Optic Nerve, by Dr. H. F. Hansell.

Report of a Case of Membranous Conjunctivitis with Systemic Complications, by Dr. H. F. Hansell.

Histories of Two Cases of Keratitis, with Abnormal Nasal Conditions as Contributing Etiological Factors, by Dr. J. N. Risley.

Exhibition of a Case of Probable Sarcoma of the Ciliary Body, by Dr. George S. Crampton.

A Case of Marked Ectasia of the Sclerotic Simulating Tumor, with Exhibition of the Specimen, by Dr. G. S. Crampton.

Exhibition of a Case of Chorioretinitis in a Patient with Diabetes and Nephritis, by Dr. W. T. Shoemaker.

Exhibition of a Patient with Central Chorioretinitis, Resembling Retinitis Circinata, Associated with Diabetes, by Dr. W. T. Shoemaker.

EDWARD A. SHUMWAY,
Clerk.

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LIST OF PAPERS: SECTION ON OTOLOGY AND LARYNGOLOGY.

December, 1907.

Dr. G. B. Wood read a paper on the Treatment of Subacute Inflammation of the Nasal Mucosa.

Dr. A. A. Bliss read a paper on Some General Considerations in Regard to Mastoid Operations.

Dr. B. H. Potts read a Report of a Case of Extradural Abscess Simulating Brain Abscess.

Dr. B. A. Randall and Dr. Butler read a Report of Four Cases of Extradural Abscess; one with Consecutive Sphenotemporal Abscess.

January, 1908.

Dr. A. W. MacCoy read a paper on Further Observations on the Value of Lactic Acid as a Remedy for Certain Conditions of the Nasal Passages.

Dr. T. E. Stelwagon (by invitation), read a paper on Demonstration of an Etherizing Cone.

Dr. C. L. Leonard read a paper on Some Applications of the Röntgen Diagnosis to Diseases of the Accessory Sinuses, with Demonstration of Plates.

Dr. S. MacCuen Smith read a paper on Primary Mastoiditis with Objective Tinnitus.

February, 1908.

Dr. J. L. Goodale, of Boston, addressed the Section on the Relation of Local Treatment to Acute Inflammation of the Throat from a Standpoint of Pathology. Dr. Kyle opened the discussion.

Dr. G. M. Marshall read a Report of an Alveolar Sarcoma Involving the Ethmoid and Sphenoid Cavities and Orbit; Operation.

March, 1908.

Dr. D. B. Kyle read a paper on Tertiary Syphilis of the Pharynx, Tonsil and Soft Palate, Epiglottis, and Vestibule of the Larynx, with Membranous Formation Simulating Vincent's Angina. Report of three cases.

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Dr. A. A. Bliss read a paper on the Position of the Patient in Operations on the Nose and Throat.

Dr. J. S. Gibb read a paper on Some Laryngeal Complications of Typhoid Fever.

April, 1908.

Dr. B. H. Potts exhibited a Case of Double Frontal Sinusitis; Operation; Recovery.

Dr. G. C. Stout read a paper on Some Unusual Mastoid Complications.

Dr. B. H. Potts read a paper on Labyrinthitis Complicating Acute Middle Ear Suppuration.

May, 1908.

Dr. Walter Roberts exhibited a Case of Pansinusitis of the Accessory Sinuses of the Nose.

Dr. G. H. Makuen read a paper on Speech as a Factor in the Diagnosis of Deafness in Young Children. Illustrative cases.

Dr. B. A. Randall read a paper on Fatal Sepsis with Trauma of the Middle Ear and Skull.

Dr. J. P. Tunis read a paper on Some Variations in the Anatomy of the Frontal Sinus, illustrated by lantern slides.

October, 1908.

Dr. F. R. Packard exhibited a patient Showing Result of a Killian Operation.

Dr. J. S. Gibb presented a Case of Suspected Sarcoma of the Nose.

Dr. D. B. Kyle read a Report of Two Interesting Cases of Elongation of the Styloid Process Involving the Tonsil.

Dr. B. A. Randall read a paper on A Word as to Otosclerosis and Cirrholysin.

Dr. G. B. Wood read a Report of a Case of Cerebellar Abscess; Operation; Exhibition of Specimen.

November, 1908.

Dr. G. B. Wood presented a Patient Showing Fracture through Alveolar Process into Antrum and Nasal Fossa.

Dr. Ralph Butler read a Report of a Case of Mucocele of the Frontal Sinus; Exhibition of Patient.

Dr. Emma E. Musson (by invitation) read a paper on Labyrinthine Deafness; Presentation of Cases.

Dr. A. A. Bliss read a paper on Some Notes on Furunculosis of the External Auditory Canal.

Dr. G. H. Makuen read a Report of a Case of Sudden Deafness in a Child of Nine Years, with Special Reference to its Effect upon the Speech.

BARTON H. POTTS,
Clerk.

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LIST OF PAPERS: SECTION ON GENERAL MEDICINE.

November, 1907.

Observations on the Venous Pulse, by Dr. George M. Piersol (by invitation).

The Proportion of Hydrochloric Acid to Pepsin in Gastric Secretion, by Dr. Joseph Sailer.

The Nervous Manifestations of Arteriosclerosis, by Dr. Alfred Stengel.

The Clinical Significance of Fecal Bacteria, with Especial Reference to Intestinal Antiseptics, by Dr. J. Dutton Steele.

December, 1907.

A Case of Primary Cancer of the Head of the Pancreas, by Dr. Herman B. Allyn.

Congenital Torticollis, a Report of a Case, by Dr. Arthur Newlin.

The Clinical Aspects of Coagulation of the Blood, by Dr. Thomas H. Boggs, of Baltimore (by invitation).

Report of a Case of Enterolithiasis Mistaken for a Floating Kidney, by Dr. J. Alison Scott.

Epididymitis and Orchitis in Typhoid Fever, with a Report of Three Cases, by Dr. E. J. G. Beardsley (by invitation).

January, 1908.

Presentation of a Patient with Stokes-Adams Disease, by Dr. B. Franklin Stahl.

Exhibition of Pulse Tracings from the Foregoing Case, by Dr. George W. Norris.

Recent Advances in the Pathological Physiology of the Heart, with Especial Reference to Arrhythmia, by Dr. Joseph Erlanger, of Madison, Wis. (by invitation).

The Clinical Study of Heart Cases, by Dr. Lewis A. Conner, of New York (by invitation).

The Use and Abuse of Digitalis and other Heart Tonics, by Dr. Theodore C. Janeway, of New York (by invitation).

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The Importance of Instruments of Precision in the Study of Heart Cases, by Dr. John H. Musser.

Remedial Measures other than Drugs in the Treatment of Heart Disease, by Dr. Hobart A. Hare.

The Extracirculatory Manifestations of Cardiac Failure, by Dr. Alfred Stengel.

Blood Pressure in Relation to Prognosis in Heart Diseases, by Dr. Joseph Sailer.

February, 1908.

Exhibition of the Skeleton of a Case of Rickets, by Dr. L. T. Donhauser (by invitation).

Report of a Case of Chronic Lymphatic Leukemia, by Dr. William Pepper.

Report of a Case of Pneumonia Complicated by Pneumococcus Arthritis, by Drs. Alfred Hand, Jr., and John H. Jopson.

Gallop Rhythm of the Heart, by Dr. G. Canby Robinson.

Report of a Case of Fibrinous Bronchitis, by Dr. E. Burvill-Holmes (by invitation).

Levulose a Test of Hepatic Function, by Dr. Edward H. Goodman.

March, 1908.

Exhibition of Two Patients having Thoracic Aneurysms, and Presentation of Specimens of a Third Case, by Dr. J. Alison Scott.

Exhibition of a Patient having an Aneurysm of the Thoracic and of the Abdominal Aorta, by Dr. George W. Norris.

The Resistance of Diabetics to Bacterial Infection, by Drs. J. C. Da Costa, Jr., and J. G. Beardsley.

A Case of Typhoid Fever ushered in by an Attack of Uremia, by Dr. R. Max Goepp.

April, 1908.

The Complications and Sequelæ of Measles, based on a Study of 460 Cases, by Dr. H. R. M. Landis.

Lobar pneumonia, a Study of 445 Cases, with Especial Reference to the Decreased Mortality since the Institution of the Fresh-air Treatment, by Dr. George W. Norris.

May, 1908.

The Anemia of Hepatic Cirrhosis Simulating the Pernicious Anemia Type, by Dr. James E. Talley.

Report of a Case of Dilatation and Hypertrophy of the Heart, with Mitral Valve Disease and Tricuspid Insufficiency, by Dr. Herman B. Allyn.

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Exhibition of Some Radiograms of the Foregoing Case, by Dr. Charles L. Leonard.

Exhibition of a Series of Cardiosphygmograms from the Same Case, taken at Intervals during the Last Two Years, by Dr. George W. Norris.

Exhibition of Some Cardiac and Pulmonary Cases of which Dr. Leonard Exhibited the Radiograms, by Dr. Albert P. Francine.

Demonstration of the Recent Advances in Röntgenography of the Heart and Lungs, by Dr. Charles L. Leonard.

DR. GEORGE W. NORRIS,
Clerk.

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LIST OF PAPERS: SECTION ON MEDICAL HISTORY.

April, 1908.

Dr. Chas. W. Burr read a paper entitled Some Remarks about Dr. Lettsom.

Dr. Joseph Sailer read a paper on the State of Medicine as Depicted by Pepys in the Reign of Charles II.

Dr. Francis R. Packard read a paper on Montaigne's Estimate of the Medical Profession

Prof. Joseph P. Remington exhibited a Pharmacopœia formerly the Property of William Penn.

November, 1908.

Dr. W. W. Keen, Remarks on the Incunabula and Other Books Purchased for the College during My Stay in Europe, and also on the Sepulchral Mural Tablets of Bologna; Exhibition of Incunabula and Photographs of the Mural Tablets.

Dr. Chas. W. Burr, Some Remarks on the Life of Jerome Cardan.

Dr. James J. Walsh, Professor of Medical History, Fordham University, School of Medicine, read a paper on Old-time Medicine and Medical Education.

WILLIAM PEPPER,
Clerk.

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